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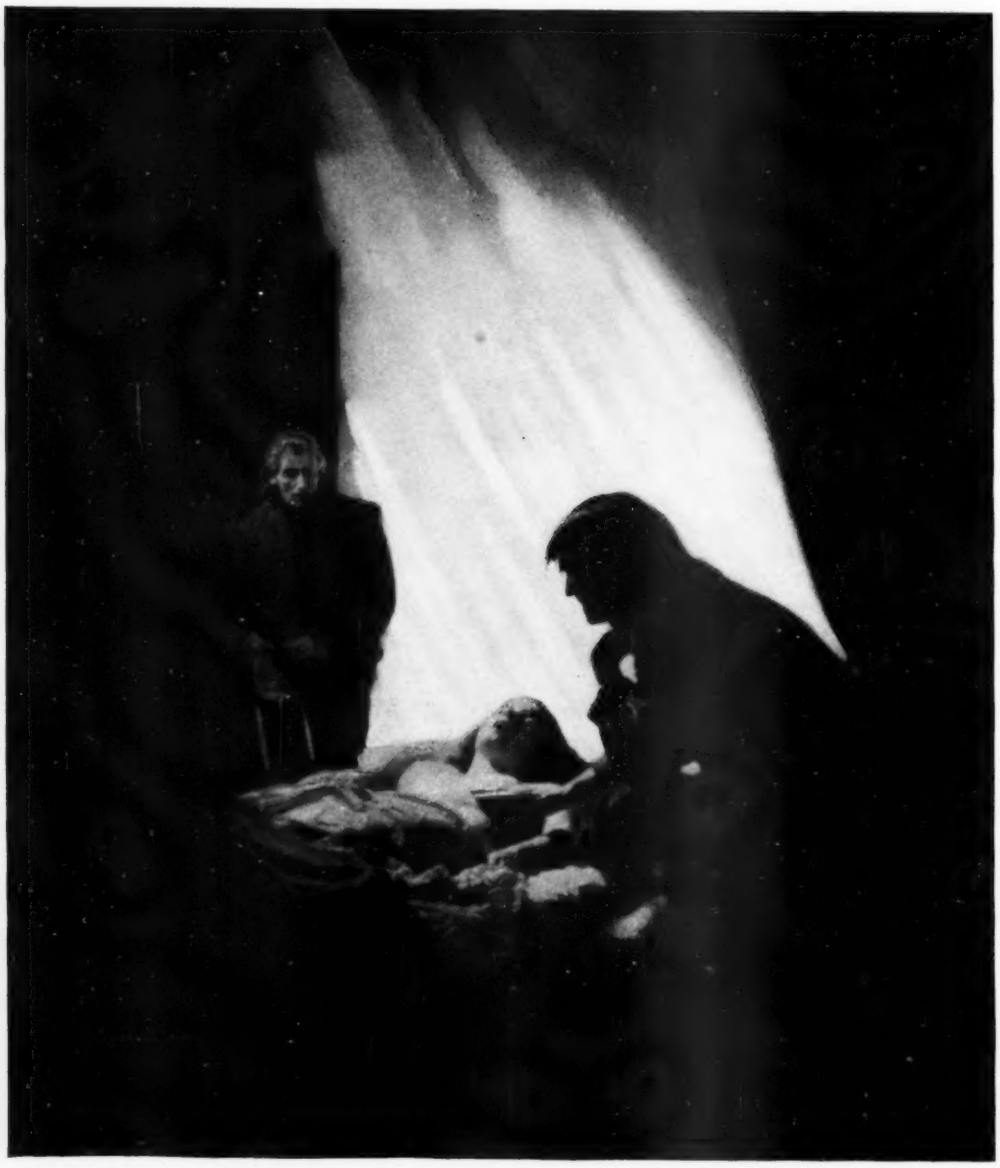

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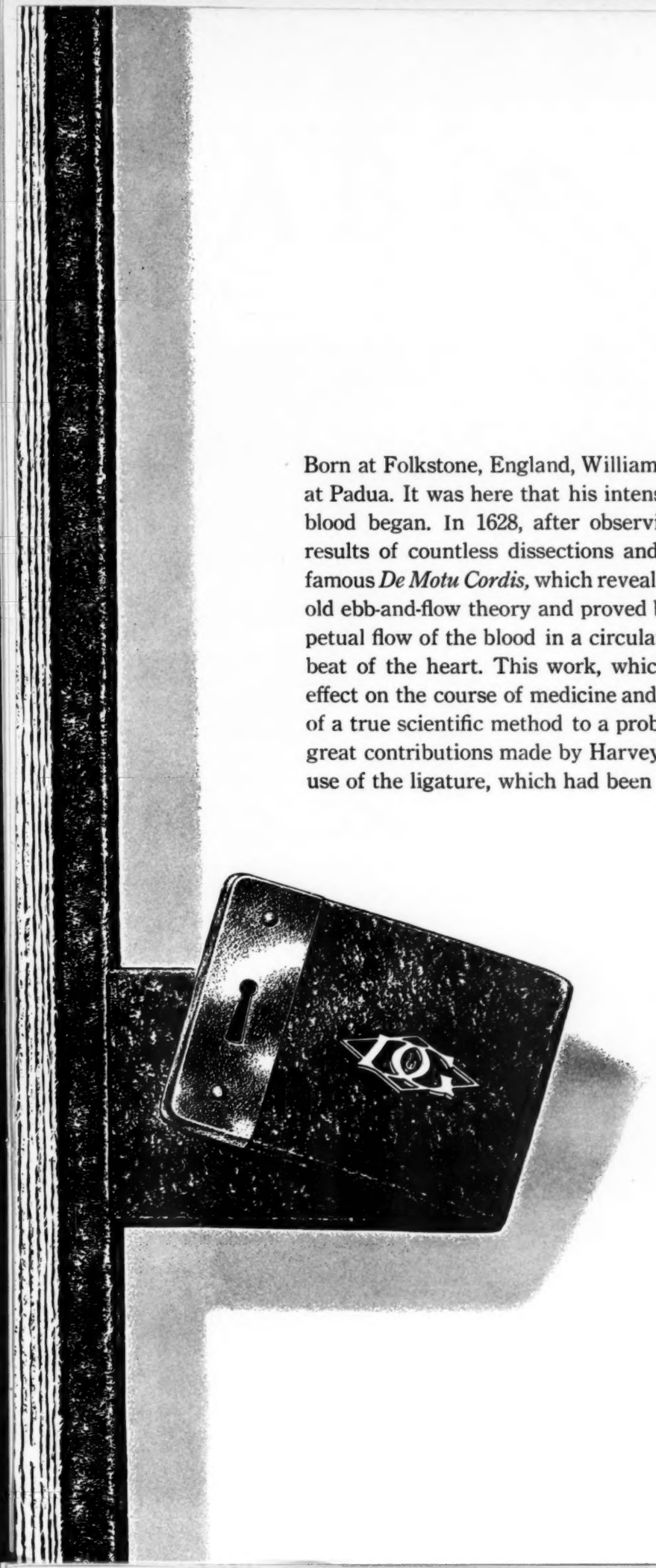

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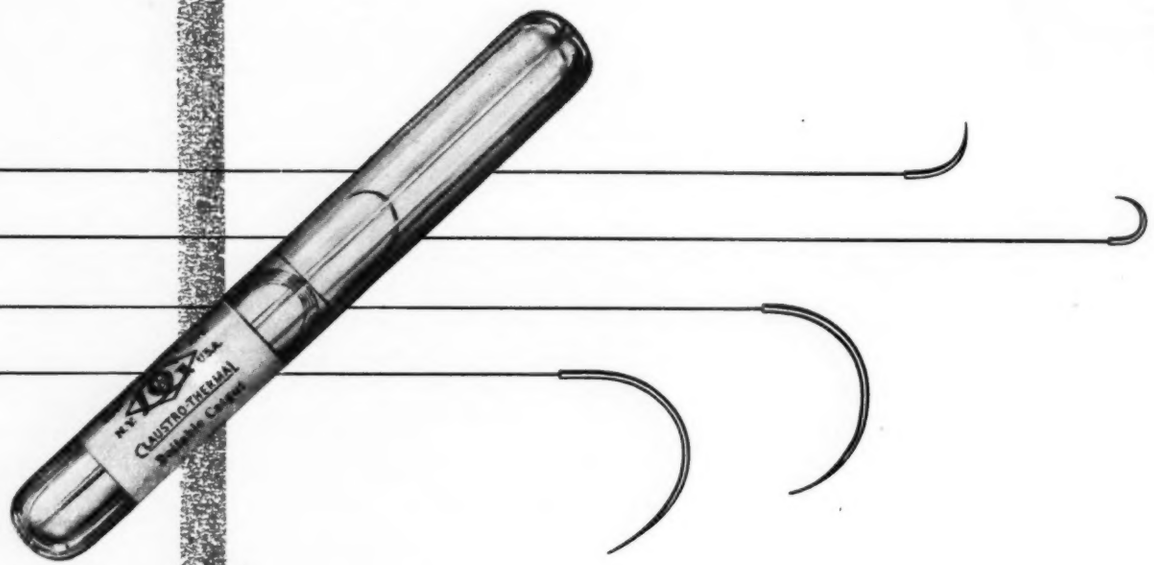

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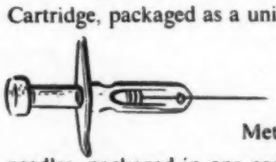

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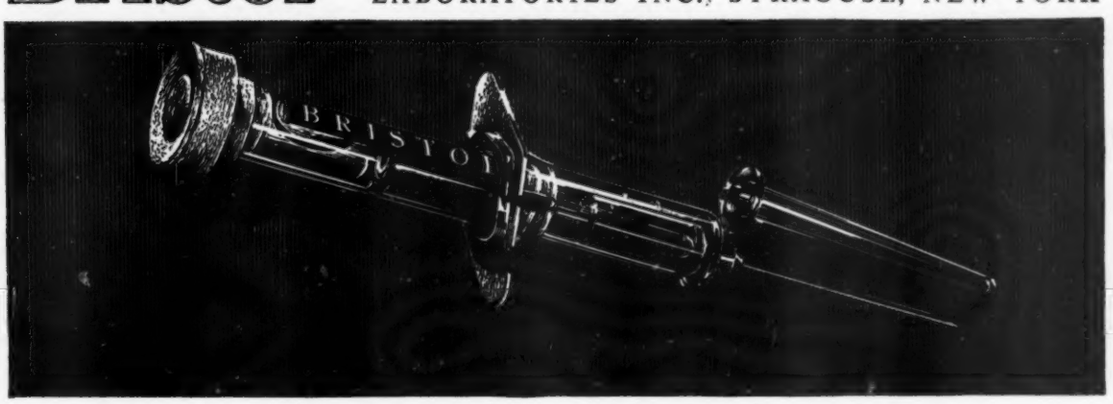

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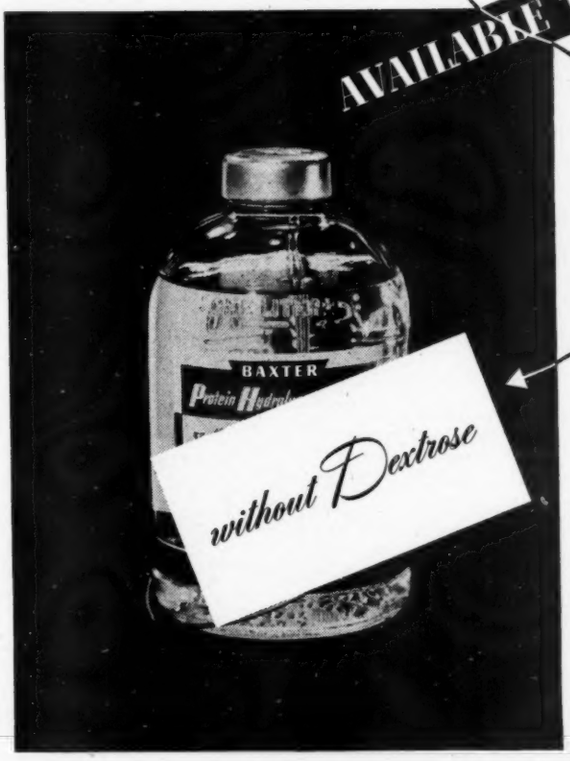

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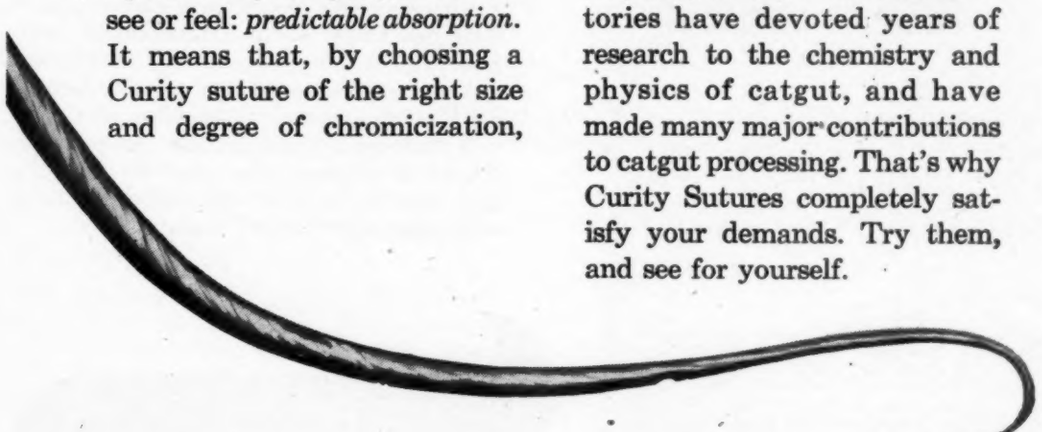

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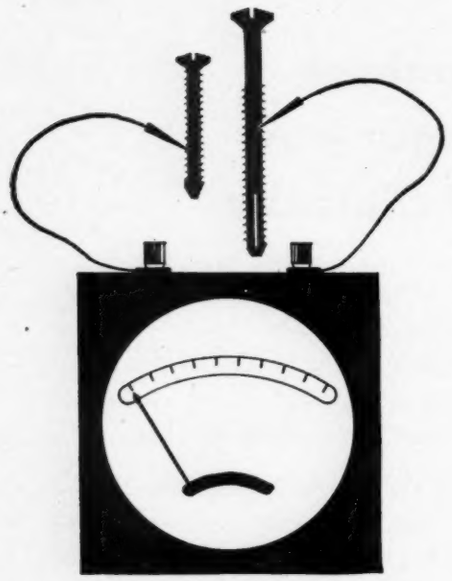

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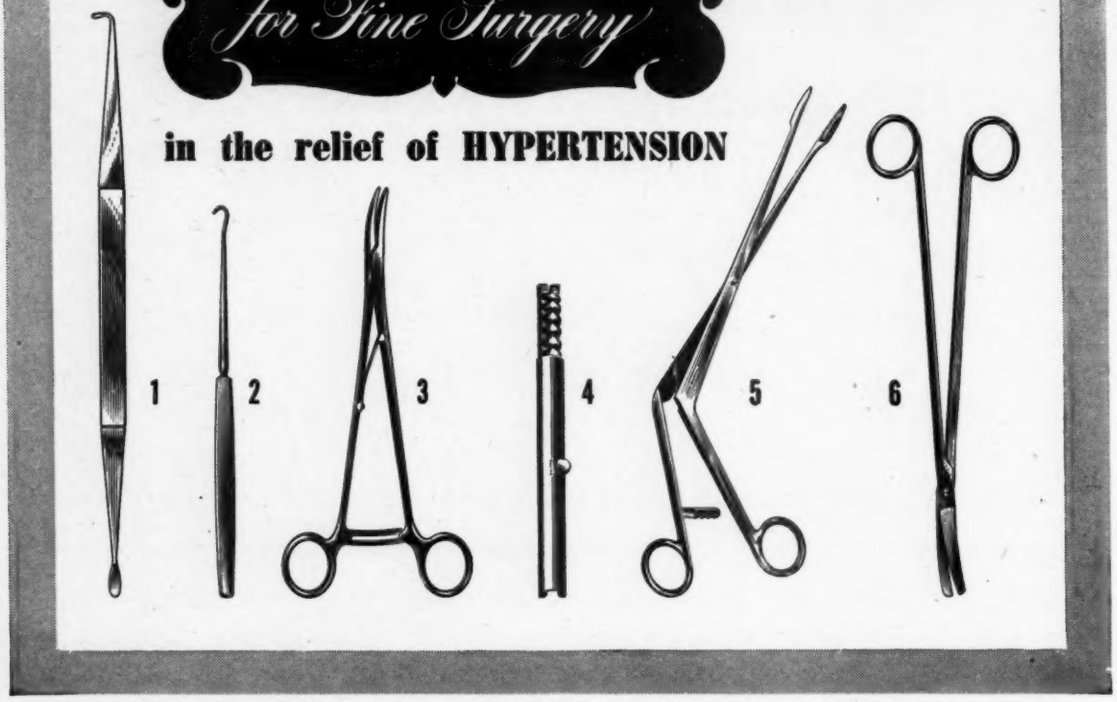

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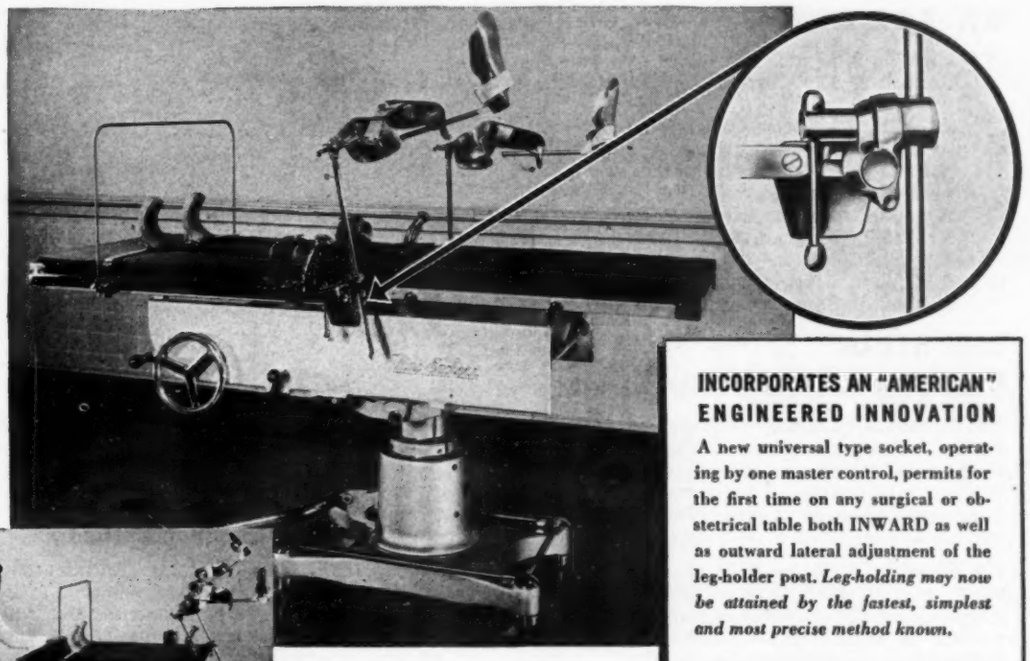

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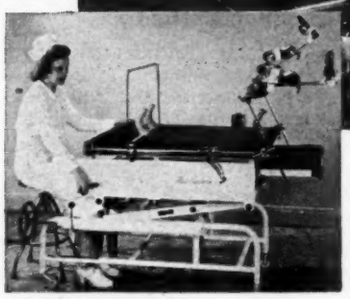

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IMPORTANT! The taking of smears, scrapings, etc. *in preparation* for staining and diagnosis is a relatively simple matter and can be done by any trained Doctor or Technician—the staining and diagnosis should be done *only by those prepared by intensive training in the technic.*

KIT FOR UTERINE CANCER DIAGNOSIS

Catalog No.			
A–2260	1000 Ayre's Cervical Biopsy Scraper. (Packed 500 per box)	per M per C	$15.00 2.00
A–2261	1 Bottle OG 6 and 1 Bottle EA 50 (two bottles)	1 each	6.00
A–2262	1 Bottle Harris Hematoxylin	each	1.25
A–2263	12 Vaginal Pipettes	dozen	7.20
A–2264	3 Aspirator bulbs	dozen	7.20
A–1460X	1 gross Rite–on slides, 3 x 1″	gross	2.75
A–1625C	3 dozen two slide mailers with envelopes	dozen	.90
A–2250	Uterine Cancer Diagnostic Outfit, including all of the above items		$36.00
A–2255	Same as above, but without stain (A–2261, A–2262)		28.75

THE STAIN

THE PAPANICOLAOU STAINS

EA 50 and OG 6 were developed as general stains for vaginal smears. They are particularly well suited for the early detection of uterine neoplasms. These stains have also been employed as useful cytological aids in certain endocrine and sterility studies.

EA 50 and OG 6 have been utilized for the study of exfoliated cells in some body fluids. Atypical cellular structures have been demonstrated in specimens obtained from urinary, parecentetic, thoracentetic, sputum, gastric, and external ulcerated sources.

These stains are to be used in combination with Harris Hematoxylin. This staining procedure results in a sharp nuclear stain, with preservation of nuclear detail and light differential coloring of the cytoplasm which preserves the translucency of the smears.

Harris Hematoxylin (Ortho Modification) was especially designed for use with EA 50 and OG 6. This hematoxylin is an excellent nuclear stain. It is previously acidified, has excellent keeping qualities and is packed ready for use.

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Seventy 2 x 2″ (35 mm.) Kodachrome Photomicrographs are now available in a series which will consist of approximately 100 photomicrographs made with the cooperation of DR. J. ERNEST AYRE, Director, Gyne–cytology Laboratory, Royal Victoria Hospital, Montreal, Canada, showing . . .

Normal Cell Patterns in Different Age Groups and
at Different Stages of the Menstrual Cycle, for . . .
A.) Reproductive Period
B.) Post–menopausal Period

Also showing . . .

SELECTIVE CYTOLOGY SCRAPINGS
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A three months combined full time refresher course consisting of attendance at clinics, witnessing operations, lectures, demonstration of cases and cadaver demonstrations; operative eye, ear, nose and throat on the cadaver; clinical and cadaver demonstrations in bronchoscopy, laryngeal surgery and surgery for facial palsy; refraction; radiology; pathology; bacteriology; embryology; physiology; neuro-anatomy; anesthesia; physical therapy; allergy; examination of patients pre-operatively and follow-up post-operatively in the wards and clinics.

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One blade of Berger clamp placed into the bowel.

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● **This instrument—designed by Dr. Louis Berger, Attending Surgeon, Gastro-intestinal Service, Jewish Hospital, Brooklyn, N. Y., and made by Weck—is of unique construction. In use its construction does not permit rotation or separation of the tips of the clamp and thus assures uniform pressure on spur. The approximation begins at the tip and thence extends to the length of the crushed portion.**

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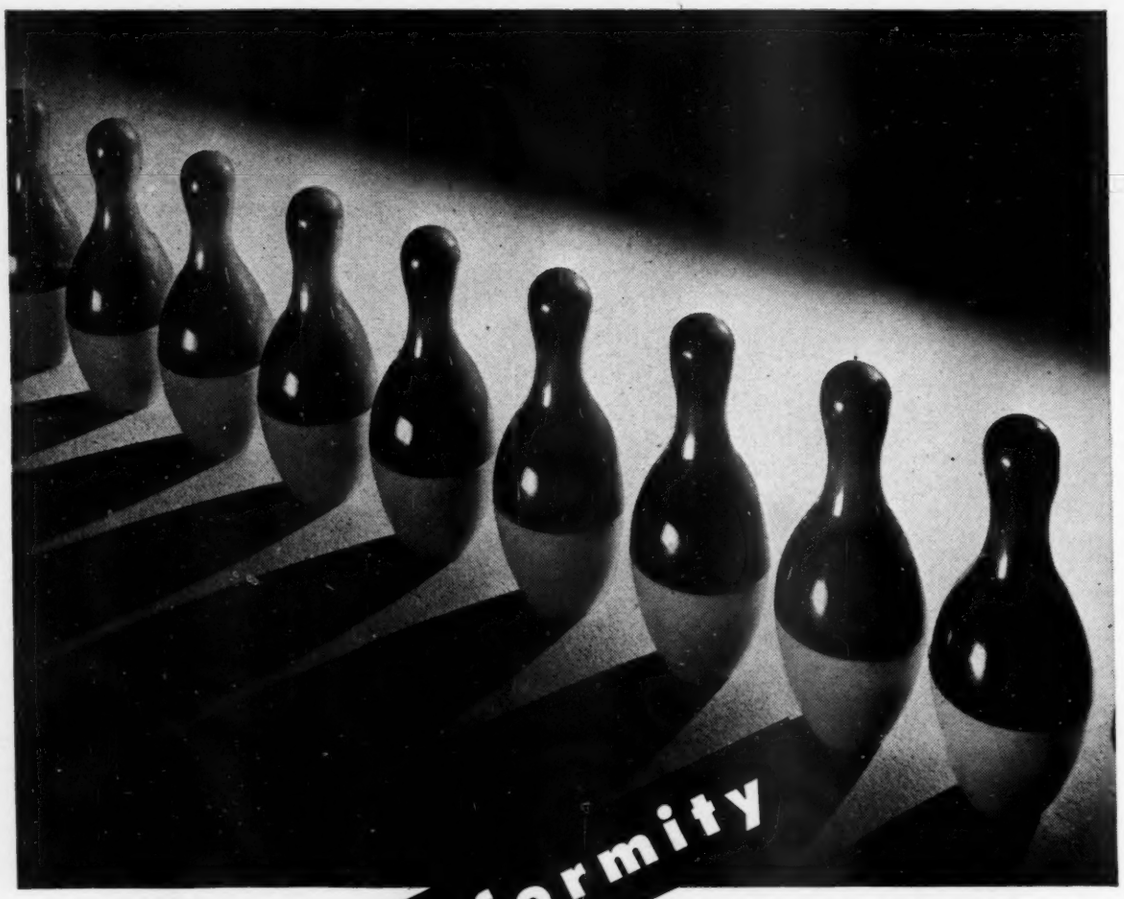

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1. Lehr, D.: Proc. Soc. Exper. Biol. & Med. 64:393, 1947.

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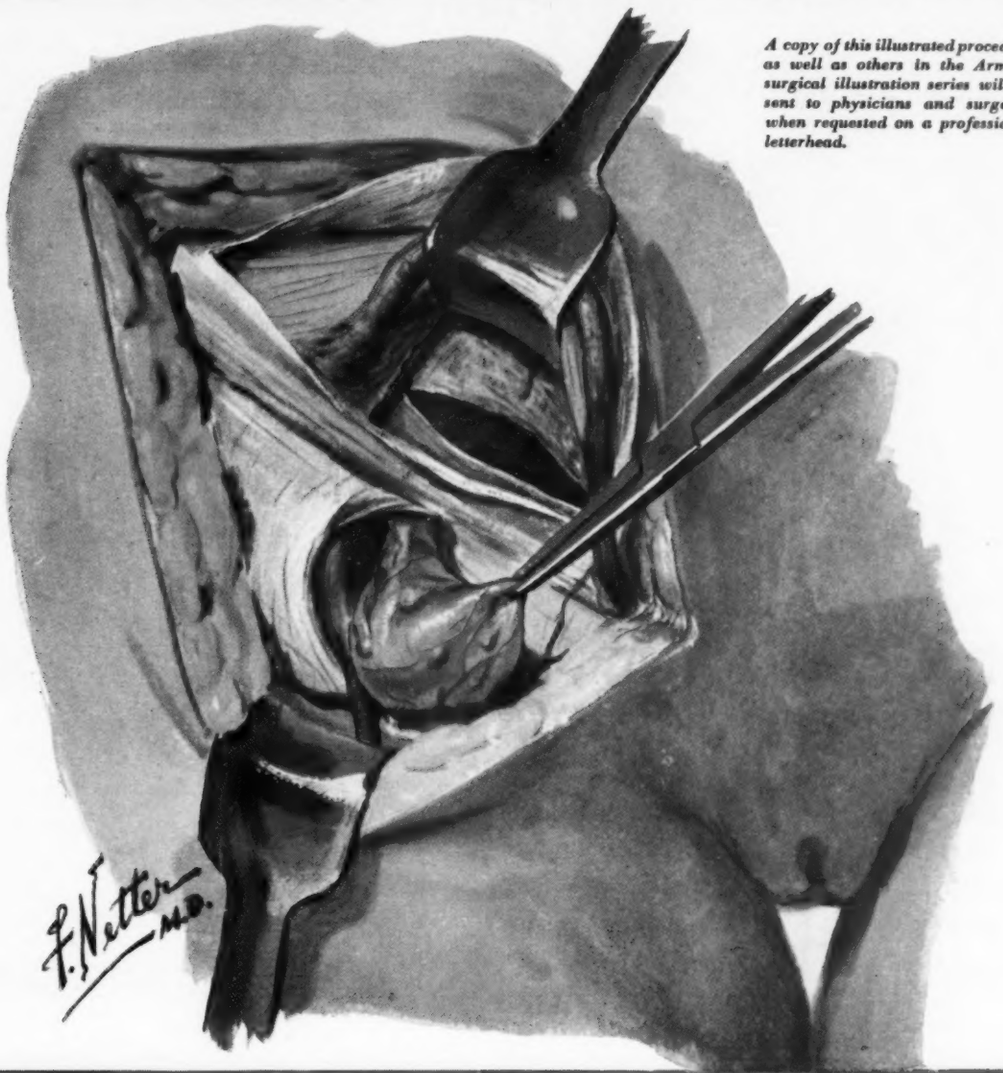
*Dodd, M. C. & Stillman, W. B.: J. Pharmacol. & Exper. Therap. *82*:11, 1944.
*Unpublished work from the Research Department of Eaton Laboratories.



A copy of this illustrated procedure as well as others in the Armour surgical illustration series will be sent to physicians and surgeons when requested on a professional letterhead.

FEMORAL HERNIA

There are numerous variations and modifications in the technique of femoral herniorrhaphy. There are those who prefer the approach from above the inguinal ligament, and those who prefer the approach from below. Some purse-string the sac, some transfix it . . . others invert it. In general, however, most surgeons adapt the procedure to fit the anatomical conditions of the individual case. The basic requirement for good results is always "sound surgery." And such surgery demands the best in suture and ligature materials. But "quality" in suture materials means more than just tensile strength, uniformity and sterility. ARMOUR SUTURES and LIGATURES have all these and something more. They are known for their excellent "working properties", their flexibility, pliability, good tying qualities. To facilitate the operation and to insure the suture line it is sound practice to specify ARMOUR SUTURE and LIGATURE MATERIALS.

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FEMORAL HERNIA

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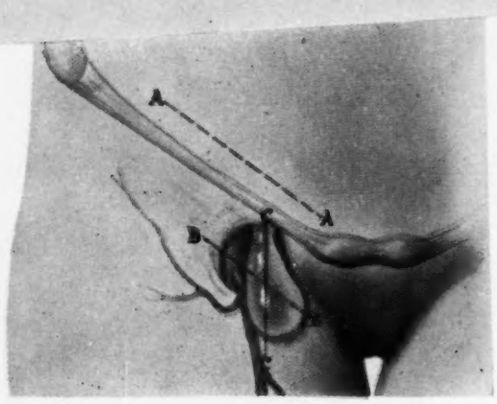
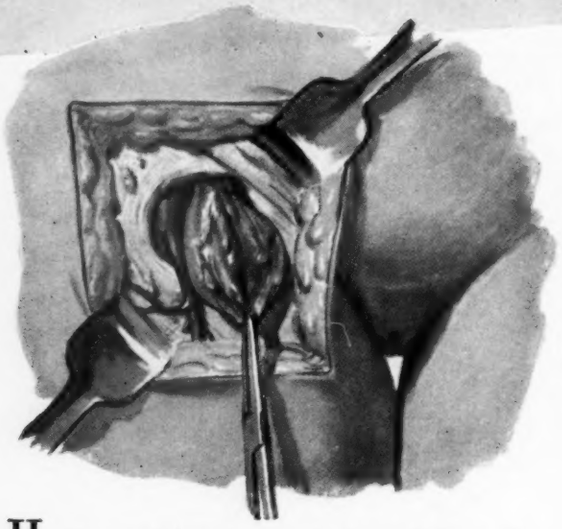
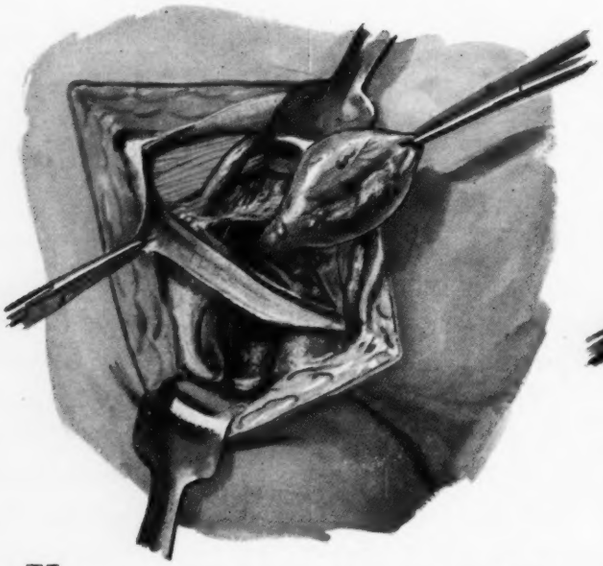


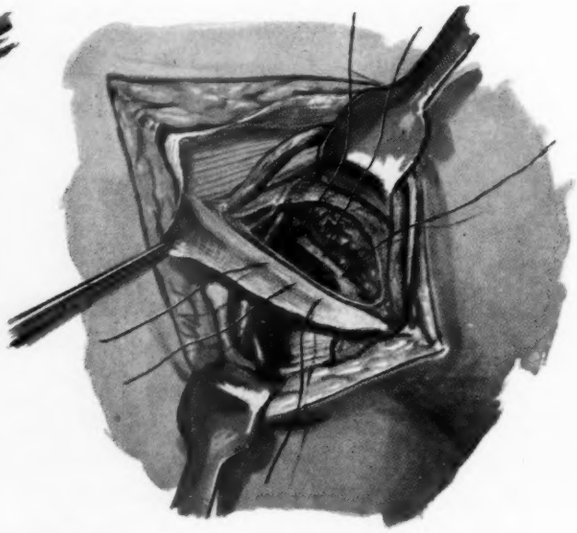
I. Topographical anatomy: The femoral hernial sac passes through the femoral canal, medial to the femoral vein, emerging through the fossa ovalis. A-A indicates the incision for the superior (inguinal) surgical approach to femoral hernioplasty. Incision B-B or C-C used for the femoral approach inferior to the inguinal ligament.



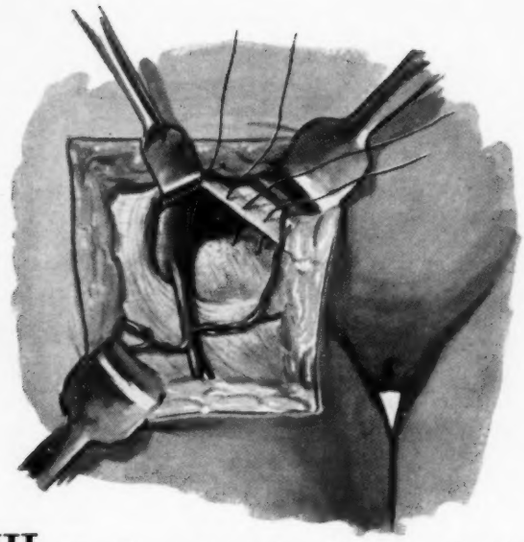
II. Femoral hernia repair from below; dissection of the hernial sac.



V. Sac has been drawn upward through the femoral canal beneath the inguinal ligament, thus converting it into a "direct" hernia. Cooper's ligament is exposed.

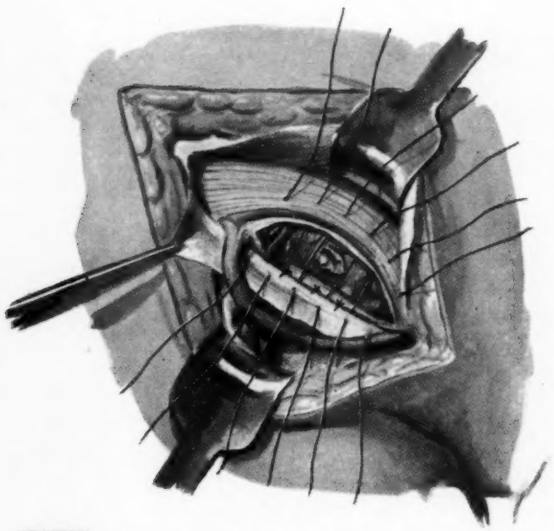


VI. Sac has been transfixed, doubly ligated and amputated. Femoral canal closed to medial border of the femoral vein by suturing the shelving margin of Poupart's ligament to Cooper's ligament.

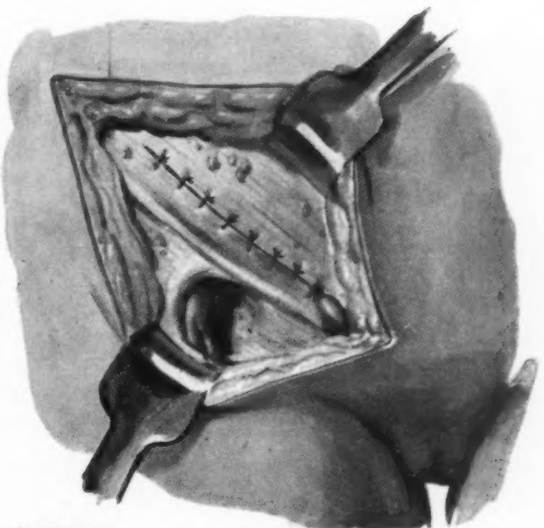


III. The femoral sac has been transfixed, doubly ligated and amputated high up in the femoral canal. Femoral canal closed to medial border of the femoral vein by means of mattress sutures approximating the inguinal ligament to the pectineus fascia.

IV. Repair of femoral hernia from above. The sac is mobilized high up in the femoral canal. Transversalis fascia incised medial to the inferior deep epigastric vessels to expose the neck of the femoral hernial sac above Poupart's inguinal ligament.



VII. Repair of the inguinal canal. Arching fibers of the internal oblique muscle, the conjoined tendon and the transversalis fascia sutured to the inguinal ligament.



VIII. External oblique aponeurosis sutured over the round ligament (or spermatic cord in the male).

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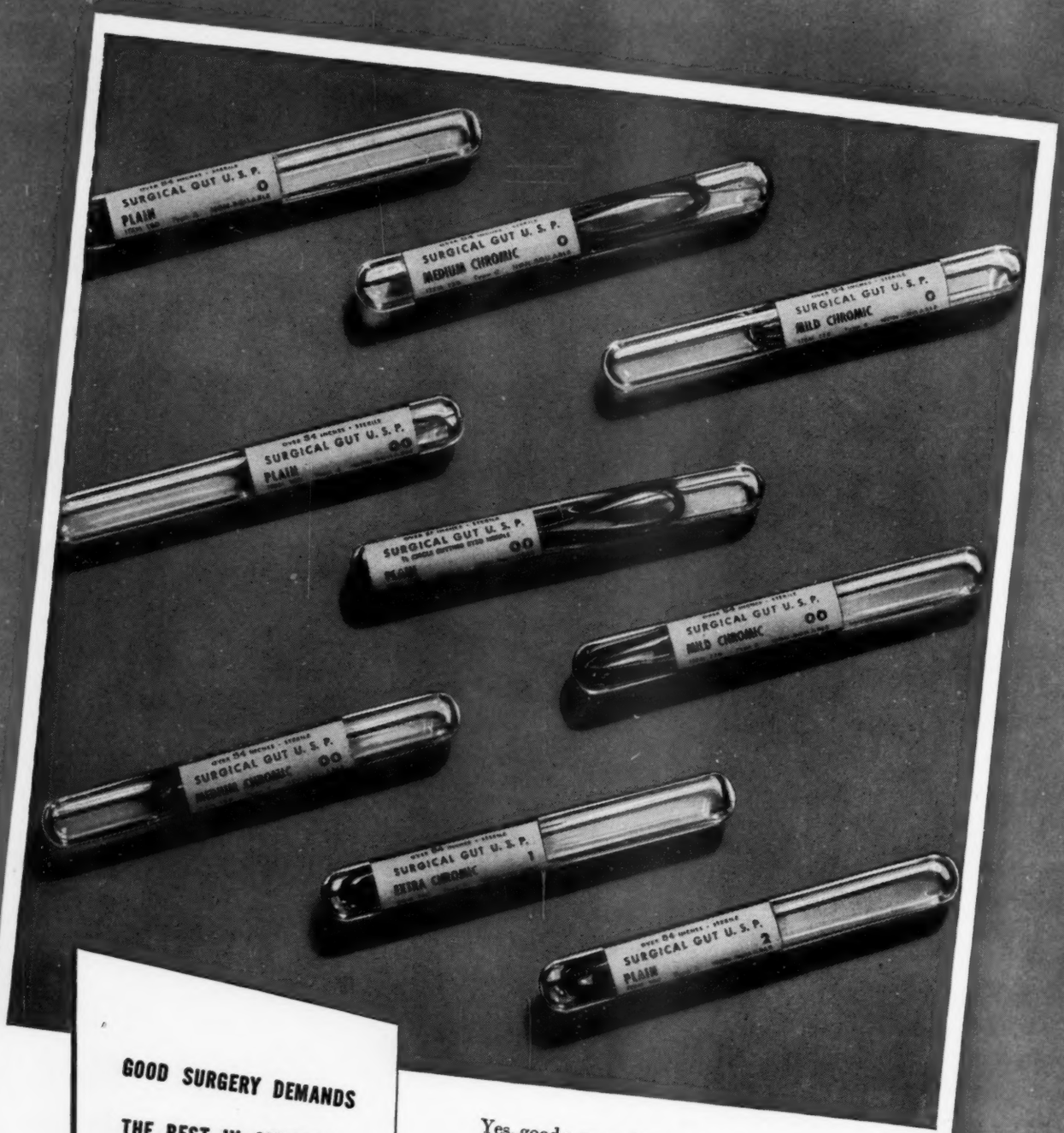

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No country is better than the nutritional state of its people. Maintaining a good nutritional state is a difficult problem unless a nutritionally adequate breakfast is eaten. Recent evidence† shows that the nutritional deficit created by a skipped or skimpy breakfast can hardly be overcome by the other two meals of the day. Hence the habit of neglecting breakfast, so widely prevalent among children of school age as well as adults, is a serious threat to the nutritional state of the nation.

A widely accepted basic pattern which forms the foundation for a nutritionally adequate breakfast consists of fruit, cereal, milk, bread and butter. It provides 611 calories and virtually all essential nutrients in substantial amounts. Furthermore, this breakfast is notably economical, and is universally available. The cereal serving—consisting of hot or ready-to-eat breakfast cereal, milk, and sugar—is an important component of this meal. It adds taste appeal and variety, and makes its own significant contribution of biologically complete protein, B-complex and other vitamins, and important minerals.

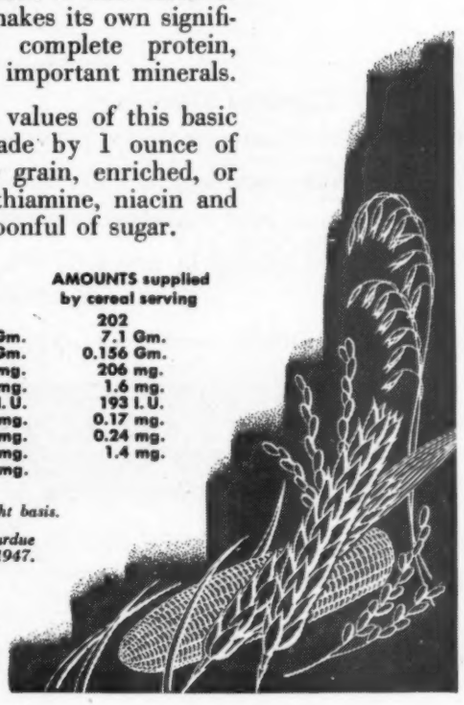
The table indicates the nutrient values of this basic breakfast and the contribution made by 1 ounce of ready-to-eat or hot cereal* (whole grain, enriched, or restored to whole grain values of thiamine, niacin and iron), 4 ounces of milk and 1 teaspoonful of sugar.

The presence of this seal indicates that all nutritional statements in this advertisement have been found acceptable by the Council on Foods and Nutrition of the American Medical Association.

BASIC BREAKFAST	TOTALS supplied by Basic Breakfast	AMOUNTS supplied by cereal serving
Orange juice, 4 oz.		
Ready-to-eat or Hot Cereal, 1 oz.;	CALORIES..... 611	202
Whole Milk, 4 oz.;	PROTEIN..... 20.7 Gm.	7.1 Gm.
Sugar, 1 teaspoon;	CALCIUM.....0.465 Gm.	0.156 Gm.
Toast (enriched, white) 2 slices;	PHOSPHORUS.... 488 mg.	206 mg.
Butter, 5 Gm. (about 1 teaspoon);	IRON..... 3 mg.	1.6 mg.
Whole Milk, 8 oz.	VITAMIN A..... 1074 I.U.	193 I.U.
	THIAMINE..... 0.52 mg.	0.17 mg.
	RIBOFLAVIN..... 0.87 mg.	0.24 mg.
	NIACIN..... 2.3 mg.	1.4 mg.
	ASCORBIC ACID.. 64.8 mg.	

**Composite average of all breakfast cereals on dry weight basis.*

†Jackson, P., and Schuck, C.: Dietary Habits of Purdue University Women, J. Home Econ. 39:334 (June) 1947.



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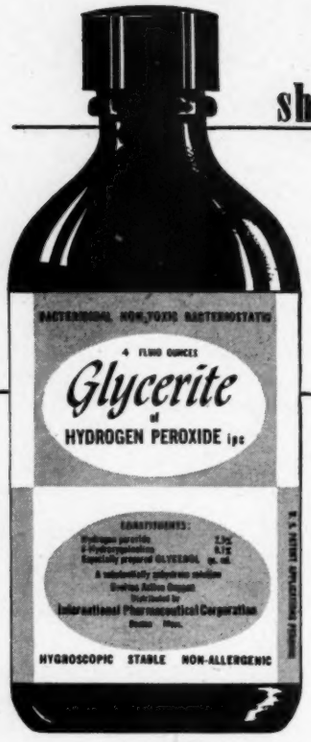

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Bibliography: *New Eng. J. Med. 234:468, 1946. J. Invest. Derm. 8:11, 1947. Science 105:312, 1947. Am. J. Surgery 74:428, 1947*

Literature on request.

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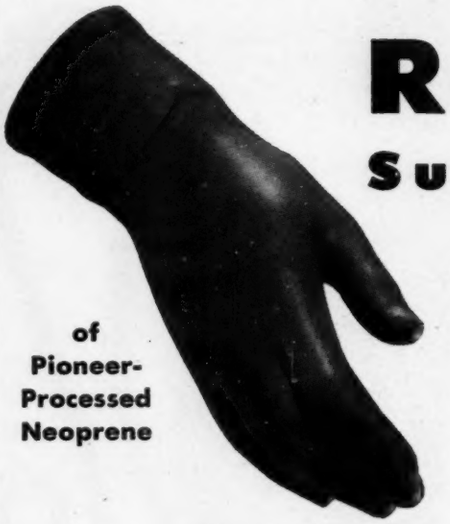

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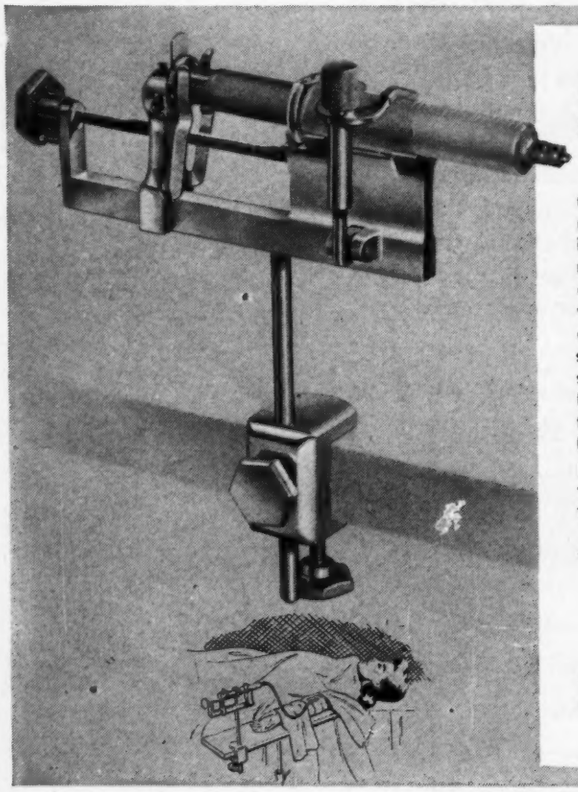

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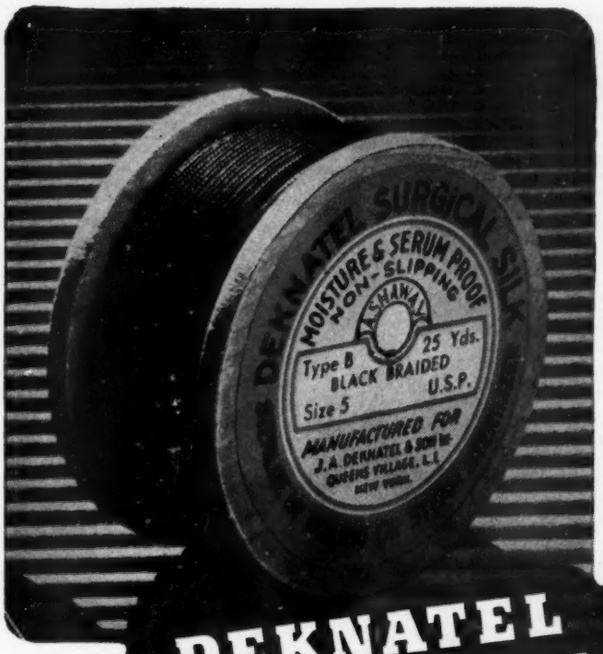

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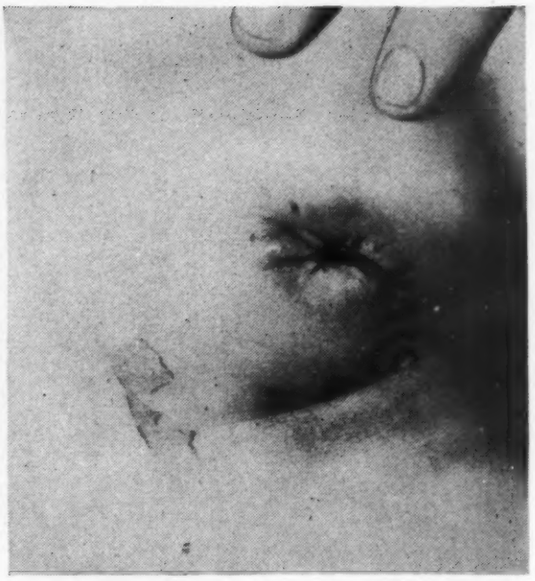

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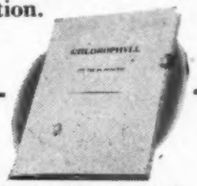

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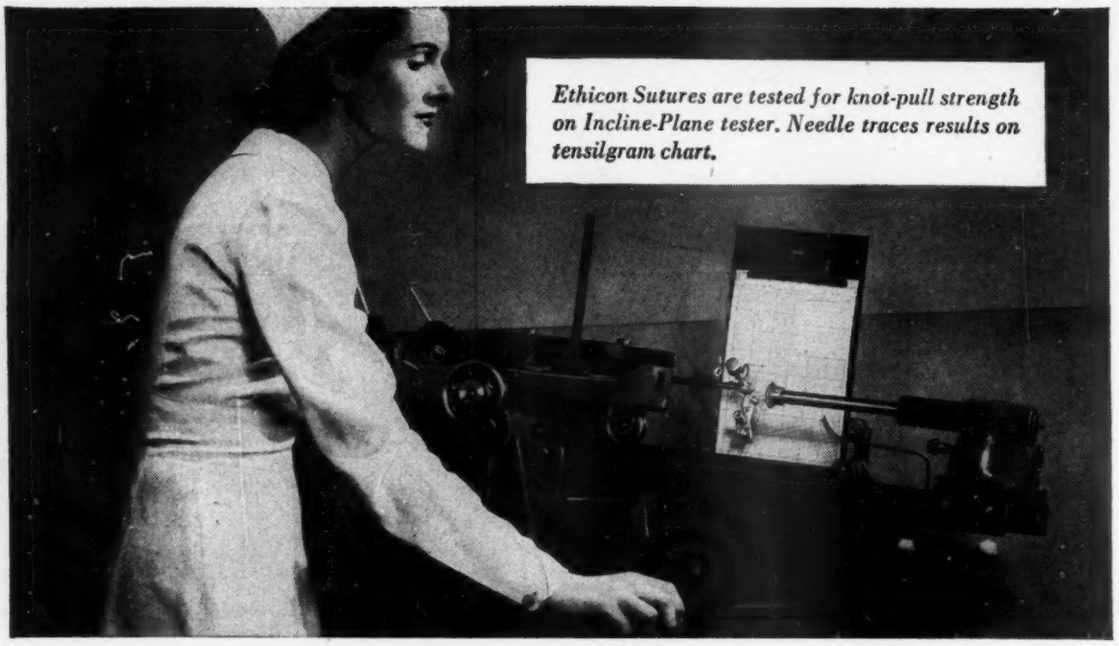

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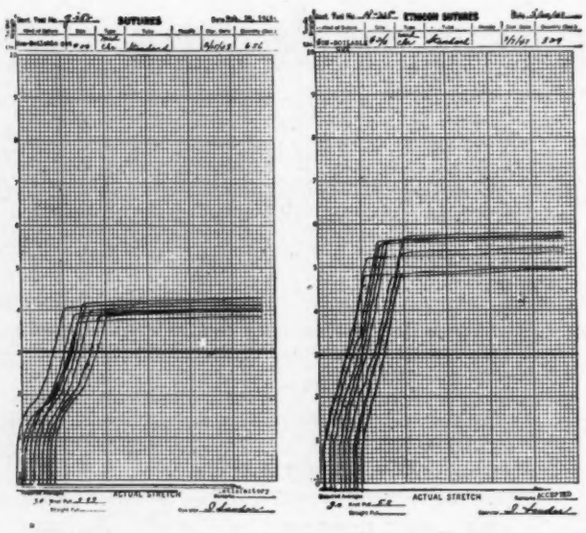

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Editorial

THE GROWTH OF NEUROSURGERY

NEUROLOGICAL surgery has come of age as a specialty in most of the large communities of the United States. This maturity has not been attained without certain vicissitudes during the adolescent stage of development. Throughout this period of growth the preeminence of Harvey Cushing as a scholar, teacher and surgeon was the guiding influence in the furtherance of high ideals in those engaged in this particular field of surgical endeavor. In addition, the teachings of a host of eminent neurologists, the ever-widening sphere of experimental investigation and the contributions of many ingenious surgeons, notably Walter Dandy, have been determinants in the establishment of the enviable standards set for this specialty. The amalgamation of these, as well as other distinctive features of neurology and surgery with particular emphasis on practical approach to diagnosis and scrupulous adherence to Halstedian surgical technic, bids fair to promote teaching that will produce more efficient neurosurgeons for the future.

The gradual formation of national, regional and local neurosurgical societies attests to the serious intent of the increasing numbers of young men entering this field. The senior organization, the Society of Neurological Surgeons, was founded in 1920 and now has sixty-three members. The Harvey Cushing Society was founded in 1931 and now has 140 members. The American Academy of Neurological Surgeons was founded in 1908 and now has forty-three members. Now a local organization, the New York Neurosurgical Society, was founded in 1946 and has twenty-seven members. In addition to these organizations the American Board of Neurological Surgery was established in 1940 and to date has certified approximately 250 candidates. Medical schools have recognized the importance of the teaching of neurosurgery and many of them have given departmental status to this specialty. It has now come to pass that in most schools, instruction concerning diagnosis and treatment of diseases of the nervous system amenable to surgical therapy is being given by the neurosurgeon. Medical graduates of the past decade should therefore be reasonably familiar with our accomplishments as well as our limitations. There are physicians, however, throughout the country who, for one reason or another, have not kept abreast of the advancements being made in this particular field; and it was with the thought that this special number would aid in the dissemination of such knowledge that these eminent authors were asked to contribute to this number.

Only pathologic or pathophysiologic states of the nervous system that lend themselves to surgical therapy have been given consideration. Other related subjects might have been included; however, it seemed best to limit controversial issues as well as the results of many interesting experimental problems.

A major portion of neurosurgical practice, namely, lesions resulting from trauma, has not been properly allocated to those most fitted to assume this responsibility, the trained neurosurgeon. The care of patients with lesions of the brain, spinal cord and peripheral nerves caused by contact trauma should be carried out by those especially trained in this field. During the recent war, a successful attempt was made to segregate the patients with such lesions and the surgical assessment and treatment of these was assigned for the most part to medical officers with neurosurgical training. These gathered about them younger officers who acquired experience and training especially concerning injuries of the nervous system. As these young medical officers adjust themselves to civil practice, the medical profession at large should recognize the value of their war-time experiences and give them an opportunity to apply this recently acquired knowledge.

While this symposium does not cover the entire field of neurosurgery, it is expected that the reader will find in these articles information of practical value and that this issue will serve as an aid in correctly advising patients regarding surgical treatment of diseases herein discussed.

I wish to extend a word of thanks to each author for his prompt acceptance of the invitation to participate in this symposium.

Jefferson Browder, M.D.

Original Articles

REHABILITATION OF VETERANS PARALYZED AS THE RESULT OF INJURY TO THE SPINAL CORD AND CAUDA EQUINA*

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Consultant in Neurosurgery at Cushing Veterans' Administration General Hospital, Framingham, Mass.; Formerly National Consultant for Paraplegia to the Veterans' Administration; Surgeon-in-Chief for Neurosurgery at the Boston City Hospital

Boston, Massachusetts

IT is well known that during and immediately after the first world war patients who had been paralyzed as the result of a wound or injury to the spinal cord and cauda equina had a minimal chance of survival and no chance whatever of rehabilitation to a point where they could again lead a normal social and work life. In the following years preceding the second world war, work with civilian prototypes of these patients, together with the development of certain technics for dealing with their peculiar problems, has made it possible to change this deplorable situation. The twenty-four-hour mortality in the author's clinic has been reduced to 8 per cent and the mortality at the end of the first week to 19 per cent. Deaths at the time of discharge from the hospital have been reduced from about 75 to 37 per cent and it is now possible virtually to eliminate all later fatalities. In addition, such patients as will cooperate and have the use of their hands and arms can be promised a normal social and work life limited only by the restrictions made necessary by their enforced use of braces and crutches. This knowledge was first available only just in time for its application to the so-called "paraplegic" soldier.[1] Because of certain characteristics peculiar to the circumstances of their wounding and immediate care, it was possible to study in more detail certain complications of these injuries and to extend the benefits gained by this knowledge to both civilian and service patients. Nutritional problems, for example, that, because of lack of material in the civilian group, could be no more than recognized as in need of treatment, were much more common and required urgent solution in service patients. New ideas and methods of therapy were quickly developed to a high point of efficiency. Bed sores, prevented in civilian patients before methods either could or must needs be devised for their surgical therapy, appeared in service patients in practically every case. They were of such enormous size that surgical closure was not only imperative as a life-saving measure but essential for any degree of rehabilitation as well. Such complications as renal and bladder stones, hypertrophy of the internal urethral sphincter and the effects of prolonged suprapubic and perineal bladder fistulas, could all be studied. Finally, the usefulness of certain surgical procedures[2,3,4] in the treatment of spasm was confirmed and the operations standardized.

* The opinions expressed herewith are those of the author and do not necessarily agree with those of the Veterans' Administration or the United States Army or Navy.

With the cessation of hostilities, the service patients were transferred to the Veterans' Administration for completion of their therapy. This group not only had to face the same medical and surgical problems that the Armed services had had to (with the exception, of course, of the immediate care of the wounded) but also was forced to assume the burden of rehabilitation as defined above. The alternative was to resign itself to the expense and trouble of providing domiciliary care for these wounded veterans for the rest of their lives. Because such rehabilitation on such a scale was virtually a new problem, certain centers were designated for the concentration and hence more efficient treatment of the "paraplegics." This paper is a brief study of the results of the first year of treatment of patients paralyzed as a result of wounds and injuries to the spinal cord and cauda equina and cared for at Cushing Veterans' Administration General Hospital, one of those centers. It covers the period from October 1, 1946, to October 1, 1947.

For comparison, certain figures in relation to this problem that were collected from five Army and Veterans' Administration paraplegic centers as of August 1, 1946, are interesting. Six hundred ninety-two cases of injury to the cord and cauda equina were analyzed. Sixty-two per cent were paraplegics in the medical sense of the term, that is, had complete motor and sensory paralysis below the level of the cord injury; 3 per cent were quadriplegic, 22 per cent had partial cord lesions and 14 per cent partial cauda lesions. Less than 4 per cent of these patients were reported as ready for discharge from the hospital at the time of the survey. Another 4 per cent were too sick for definitive treatment. Twenty-seven per cent were still in need of neurosurgical operations, 62 per cent were not yet out of bed and 89 per cent were not yet out of wheelchairs. (Table 1.) Even though the survey did not include all the "paraplegics," I believe that the sampling was done in such a way as to approximate the general picture. Furthermore, in general, because of the time of the survey and the sampling, it can be assumed that this is a reasonably accurate picture of the cord injury problem as it was turned over to the Veterans' Administration from the Army. Table II shows the comparative figures at Cushing General Hospital, one of the centers in-

TABLE I
SURVEYED PATIENTS AUGUST, 1946

	All Cases		Percentage of Patients				
	No.	Percentage Distribution	Too Sick for Definitive Therapy	Still in Need of Neurosurgical Operations	Not Yet Out of Bed	Not Yet Out of a Wheel-chair	Ready for Discharge
All injuries	678	100	4	27	62	89	3+
Paraplegics	418	62	3	30	70	95	0+
Quadriplegics	18	3	18	0	0	0	0
Partial cord injuries	121	22	6	26	52	83	7
Sacral segment injuries	22	0	5	11	34	79	0
Partial cauda equina injuries	93	14	1	13	44	70	14

TABLE II
CUSHING GENERAL HOSPITAL AUGUST, 1946

	All Cases		Percentage of Patients				
	No.	Percentage Distribution	Too Sick for Definitive Therapy	Still in Need of Neurosurgical Operations	Not Yet Out of Bed	Not Yet Out of a Wheel-chair	Ready for Discharge
All injuries	678	100	4	27	62	89	3+
Paraplegics	84	63	0	6	56	90	0+
Quadriplegics	0	0	0	0	0	0	0
Partial cord injuries	25	21	0	21	42	59	33
Sacral segment injuries	5	0	0	0	0	0	0
Partial cauda equina injuries	46	16	0	5	23	32	36
All injuries Cushing	135	100	0	10	46	76	13

cluded in the survey, two months before the Veterans' Administration took charge. As will be noted, the figures are better in many respects than those of the entire series. This is directly traceable to the cooperative attitudes of the then Commanding Officer, Colonel Floyd V. Kilgore and his chief of the surgical service, Colonel Horatio Rogers, in having facilitated the establishment of a therapeutic program that had already been well tested in the neurosurgical department of the Boston City Hospital. It was carried out by a group of what amounted to volunteer medical officers who had had specialized training in neurologic, plastic and genitourinary surgery. Their work was coordinated and directed by a single head. This program was started in April, 1946, and by August of that year a situation that had been virtually hopeless at the start, had been changed into one that reflected tremeneous credit on the hospital and its staff. This same group of army officers augmented by two others as well as two naval officers volunteered to continue their services after the Veterans' Administration took over and are only now—after one year and four months—beginning to be replaced.

Between August 1, and October 1, 1946, the "paraplegic" census at Cushing General Hospital rose from 135 to 148. The latter was the number of patients actually on the Paraplegic Service when the Veterans' Administration took over. During the following year from October 1, 1946, to October 1, 1947, there were sixty-four new admissions to this service. Thus the total number of patients treated for injury to the spinal cord and cauda equina during the first year of the Veterans' Administration operation was 212.

There were also forty-three readmissions. Eighteen of these patients returned voluntarily for checking at various periods following their discharge. Two came back after a C.D.D. discharge from the army and three others were pursuaded to return for further necessary treatment after leaving at their own request. Five others originally left against medical advice. These have since returned for further treatment. Only seven of the readmitted patients had been carried as A.W.O.L. in that they were given leave to go home for a period and failed to come back when their leave was up. Two of the seven have since returned and have been finally discharged for disciplinary reasons; one returned and subsequently left against medical advice; one was A.W.O.L. and returned and left a second time A.W.O.L.; one returned and was then given a medical discharge; two have returned and are still under treatment.

Exclusive of readmissions, there have been ninety-seven discharges during the year and three others have completed their training and are still in the hospital only because of non-medical reasons. Fourteen have completed their ambulation training but need further medical care. Therefore, nearly one-half (47 per cent) have been or are about to be discharged living and able to take their place in the community and 54 per cent have been taught to walk in the first year of the Veterans' Administration operation.

The 112 patients that are still receiving treatment are the group that have presented all along the more difficult therapeutic problems. They include the quadriplegics (fifteen), those with intractable bed sores (forty-two), major genitourinary tract disturbance (fifteen), complicating osteomyelitis (two), a demonstrated need for neurosurgical (twelve) or orthopedic (six) operations that were not apparent until ambulation training had reached an advanced point, spasms (sixteen), amputations (three), disabling fractures (six), incidental neurologic disease (three), fear (one), laziness (six) and an uncooperative attitude (seven). Only sixteen of this group have progressed beyond a wheel-chair existence. Seventy-four are being given primary ambulation training (fifty-eight) or are still bed-ridden (sixteen). Eight are out on leave.

Two patients died before discharge. One had a transection of his lumbosacral cord and died of uremia almost two years after

injury and seven months after admission to the Cushing Veterans' Administration General Hospital. He had renal infection and stones as well as bilateral nephrostomies. The second had his cauda equina partially destroyed. He had bilateral renal stones and infection, a scrotal urinary fistula and a colostomy. He died following a kidney operation two years and five months after injury and seven months after admission to Cushing Veterans' Administration General Hospital. This mortality rate of 0.9 per cent while satisfactory from the point of view of the care given at the hospital is actually of little value from the larger point of view of the mortality as a whole in this type of case.

ANALYSIS OF THE DISCHARGED PATIENT'S CASE HISTORIES

An analysis of the case histories of the 100 patients that have completed their medical treatment and have either been or

TABLE III
CLASSIFICATION ACCORDING TO LEVEL

	Total Cases		Cervical			Thoracic			Lumbosacral			Caudal			
	Total Cases	Percentage	Transection	Partial	Radicular	Transection	Partial	Radicular	Transection	Partial	Radicular	Transection or Total Destruction	Partial	Radicular	No Note
Service cases	100	100	2	6	0	26	4	0	19	15	0	4	21	0	3
Civilian cases	313	100	29	119	9	42	37	0	6	25	0	0	44	2	0

are ready to be discharged, brings to light certain interesting data that should not be overlooked. To emphasize its significance, I have compared it with similar data obtained from an analysis of a personal series of 313 civilian cord and cauda equina injuries. (Table III.) The two series offer no fundamental differences although they do vary in certain details. In the service group the distribution of cases is preponderantly on the side of transection (Table IV), while the largest civilian group is found in the partial cord injury category. Virtually no patients were seen at Cushing Veterans' Administration Hospital in the months immediately following their injury. (Table V.) This is a complete reversal of the civilian situation and, as will be pointed out, may have an important bearing on the

TABLE IV
CLASSIFICATION ACCORDING TO PATHOLOGY

	All Patients		Transection of Cord		Partial Cord Injury		Cauda Equina Injury		Radicular Injuries	
	Total Cases	Percentage	No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage
Service cases	100	100	47	47	25	25	28	28	0	0
Civilian cases	313	100	77	25	181	60	44	14	9	4

occurrence of certain signs and symptoms that may be wrongly thought to be characteristic of cord and cauda injuries as a whole.

TABLE V
LENGTH OF TIME BETWEEN INJURY AND ADMISSION

		Less than 24 hr.	1st day	2nd through 6th day	2nd week through 1st month	2nd through 6th month	7th through 12th month	1 to 2 yr. after	2 to 3 yr. after	3 to 5 yr. after	5 to 10 yr. after	10 to 20 yr. after	Over 20 yr. after	Unknown	Totals
Cervical	Service	0	0	0	0	3	0	2	2	1	0	0	0	0	8
	Civilian	83	31	16	9	6	4	2	0	0	0	0	0	2	153
Thoracic and Lumbosacral	Service	0	0	0	0	21	4	17	14	2	1	0	0	1	60
	Civilian	37	5	10	10	7	4	8	2	6	0	1	2	1	93
Caudal	Service	0	0	0	0	7	3	12	3	0	0	0	0	1	26
	Civilian	14	1	3	4	3	5	1	2	1	6	2	1	0	43
Epidural Abscess, etc.	Service	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Civilian	0	2	4	7	3	0	2	0	0	2	1	0	0	21
Totals	Service	0	0	0	0	31	7	31	19	3	1	0	0	2	94
	Civilian	134	39	33	30	19	13	13	4	7	8	4	3	3	310

Bed Sores. Perhaps the most important and certainly the most frequent reason for prolonged hospitalization of the so-called "paraplegic" veterans is the bed or pressure sore. Fifty-three per cent of the en-

tire group of 212 patients had or have open bed sores that required active therapy. Forty-one per cent of the patients still in the hospital are there in part or wholly because of their bed sores, while 69 per cent of the patients that have been discharged had bed sores treated during their stay, 31 per cent being multiple. Many of the others had scars of sores that had healed before their arrival at Cushing. In comparison, it should be noted that only eighty-nine or 28 per cent of the 313 civilian cases had bed sores. This includes all cases regardless of length of stay in the hospital and so may be slightly biased toward the low side. If only the 204 patients that were discharged alive from the hospital are considered, the number having bed sores is reduced to sixty but the incidence remains the same—29 per cent. If the twenty-four patients that died during the first twenty-four hours after injury are eliminated as not having lived long enough to develop sores, the incidence in the remaining 289 was 31 per cent, a figure less than one-half of the discharged service group. Emphasis has been placed upon this comparison because in ideally handled cord and cauda equina injuries, bed sores should never occur. This was pointed out in 1940[5,6] and further experience merely confirms this statement.

The causes of bed sores are two: failure to restrict weight-bearing on any given bony prominence to not longer than two hours at a time and failure to maintain the patient in a constantly dry bed. Once bed sores are started they are incurable in the presence of involuntary muscular spasms, hypoproteinemia, malnutrition and anemia. The most prolific source of trouble and a virtually certain cause of bed sores in the "paraplegic" is plaster of paris dressings and of all others, plaster of paris body casts are the most harmful. With a body cast in place it is impossible to move the patient and relieve the local pressure its presence produces. Moving the patient and the cast does no good. I have been unable to determine the number of "paraplegic" soldiers that were put into plaster of paris jackets preparatory to transport to this country from overseas but certainly the figure is large. This, like the universal use of suprapubic bladder-drainage, was considered to be justified by reason of the uncertainties of transportation and because of the difficulty of ascertaining the extent and need for protection of the bony injury in any single instance. No criticism can be made of this decision but it is profitable to consider what it leads to and whether in the future it can be altered for the greater ultimate benefit of similar patients. That this has its practical aspect is evident from the comparative figures cited above. The low incidence of bed sores in the civilian group is attributable to the fact that plaster of paris was never used and because for the last several years turning every two hours, dietary measures and prevention of maceration of the skin of the back by proper nursing and the use of tidal drainage have all been rigorously adhered to.

TABLE VI
TYPE OF BONY INJURY

		No. of Cases of				
	All Cases	Transection	Partial Cord Injuries	Cauda Equina Injuries	Radiculitis Only
Major Service	43	19	13	11	0
Major Civilian	214	51	128	28	7
Minor Service	12	8	2	2	0
Minor Civilian	16	3	10	1	2
None Service	19	9	5	5	0
None Civilian	76	19	41	14	2
Total of Above Service	74	36	20	18	0
Total of Above Civilian	306	73	179	43	11
No Note Service	26	11	5	10	0
No Note Civilian	7	4	2	1	0
Totals Service	100	47	25	28	0
Totals Civilian	313	77	181	44	11

The use of plaster of paris casts is justified, *if at all*, by the necessity of splinting the injury to the vertebral column. My experience leads me to believe that this justification is present only under combat conditions, and that in the absence of such

conditions casts are unnecessary. A comparative analysis of the appropriate figures in these two series of patients supports this contention. The relative occurrence rate of a significant bony injury is 75 per cent in the civilian and 74 per cent in the service

TABLE VII
RELATIONSHIP OF BED SORE TO BONE INJURY

	Bed Sores Removed with a					
	Major Bone Injury		Minor Bone Injury		No Bone Injury	
	No.	Percentage	No.	Percentage	No.	Percentage
Service Cases.....	29	41	9	13	13	19
Civilian Cases.....	62	20	3	1	20	7

group, the significant difference being found in the severity of the injury. In the civilian group 70 per cent had major and 5 per cent minor bony injuries, while in the service group only 57 per cent had major and 17 per cent minor bone injuries. (Table VI.) It is apparent (Table VII) that not only were bed sores more than twice as frequent in association with bone injuries in the service groups but were more than two and one-half times as frequent in the absence of any bone injury whatsoever. Since the therapy has been virtually the same in both series, once the patient reached his final hospitalization, this significant difference and the resultant delay in rehabilitation must be ascribed to the methods used in the services for treatment prior to that time, and speaks strongly against the use of plaster of paris or other similar methods of immobilization in patients with spinal cord and cauda equina injuries.

Genitourinary Tract Disease and Injury: Renal and Bladder Stones. The second most disabling complication is disease and injury to the genitourinary tract. It is impossible to deal with all phases of this major problem but certain aspects may be briefly touched upon. The occurrence of renal and bladder calculi has been extremely frequent among the "paraplegics" in the services. Many papers have been written on the subject[7–12] and there can be no doubt but that it has constituted a great hindrance to the rehabilitation and sometimes to the saving of the life of these patients. In this group of 100 patients, fifteen had bladder and fourteen kidney or ureteral stones, an incidence of 29 per cent. In the civilian group there were eight bladder stones and three renal stones, an incidence of 4 per cent if only the 252 patients that lived eight days or longer after injury, that is, long enough to develop stones theoretically, are considered. Five of the eleven patients that had stones had long histories of treatment elsewhere with formation of stones during that time, thus reducing the actual incidence of stones under treatment by tidal drainage to six in 252 patients, or 2 per cent. This is too great a discrepancy to be passed over lightly especially when the virtual parallellism of the two series is taken into consideration. Special diets,[13] elevation of the head, etc.,[8,12] have been proposed as being necessary for the elimination of genitourinary tract calculi. I can only state that no such measures were even considered, much less carried out, in this civilian group of cases. The most important difference between the two series of cases, however, is, as pointed out, one of the time of application of what I have come to regard as the essential minimal therapy of the genitourinary tract in cord injury patients. This requires, among other things, the use of tidal drainage with its maintenance of normal anatomic and functional states of the bladder and its prevention of major infection of the genitourinary tract.[14] A constantly dry bed and the ingestion of adequate amounts of water are also essential.

Practically all of these requirements were unfortunately disregarded or omitted of necessity during the first stages of treatment of the service "paraplegics." Most

had and all were supposed to have had a suprapubic bladder fistula produced. This violates the anatomic and functional normalcy of the bladder. In addition, after the suprapubic fistula was closed, tidal drainage was often not used for a considerable period of time. As a result the bladder either shrank and became overactive or stretched and become virtually atonic. Infection involving both the bladder and kidneys was inevitable under such conditions. Because the infection was subclinical did not mean it was any the less lethal in so far as serving as a groundwork in which stones could and would form.[15] To illustrate the importance of this there were twenty-five major infectious processes affecting the kidneys, ureters and bladder in the service and only nine in the civilian group. The comparative percentages are 25 and 4. It seems reasonable to assume in the light of these figures that the presence of infection is a major and may be the determining factor in the production of renal and bladder stones in "paraplegics." The custom of draining their bladders by a suprapubic fistula certainly does not detract from this assumption. Albright et al.[15] and Albright in a personal communication states that among other causes of genitourinary tract calculi common and important ones are the development of a nidus without local stasis at any point of injury to the mucous membrane, the persistence of an alkaline urine and tubular damage without associated glomerular damage. This last leads to hypercalcuria and is caused by chronic genitourinary tract infection which is usually staphylococcal and often subclinical. It would appear then that there is greater chance of preventing the formation of renal calculi in "paraplegics" by concentrating on prevention and control of genitourinary tract infection and subsidiary conditions such as an alkaline urine than by manipulation of diets and the use of other less direct procedures. Experience has demonstrated that the prompt and continued use of tidal drainage[16] is the best method of doing this.

Resection of the Internal Urethral Sphincter. Emmett[17] and Thompson[18] have advocated resection of the internal urethral sphincter in patients with cord injuries, tabes dorsalis and a theoretic congenital thickening of the internal sphincter that causes so-called "unexplained cord bladder." It is clear that they have practiced this operation only on patients that have had their genitourinary tract lesions for a long time. It is also clear that their experience has not included patients with cord injuries during their earlier stages and they are therefore not familiar with the stages of recovery that a bladder passes through if properly treated. By implication and omission they would lead the careless reader to conclude that resection of the internal urethral sphinter is appropriate and therapeutic in all patients with cord injury regardless of the length of time that has elapsed since injury and regardless of the treatment they have received for their bladder. There is also a widespread belief among surgeons and patients to this same effect and some tendency to regard this operation as a panacea in the care of the genitourinary problems in "paraplegics." The operation is undoubtedly essential in the demonstrated presence of hypertrophy or other similar affection of the internal sphincter if the latter prevents evacuation of the bladder at every emptying contraction. The operation is not without danger, however, in that if too much tissue is removed the patient becomes permanently incontinent, a condition which cannot then be controlled even by an inlying catheter. The procedure also appears to be unduly risky and probably unsuitable for women because of the peculiar anatomic distribution of the sphincteric muscle fibers. It is contraindicated in the face of a flaccid or spastic external sphincter.

I believe that the need for this resection can virtually be eliminated if proper care is provided for the bladder from the start. A comparison of the appropriate data in both of the present series of cases will bear this

out. Nine of the service patients had their internal sphincters resected. Six had transected cords and three cauda equina injuries. Four of the transected cord cases had "spastic" and two hypertrophied internal sphincters. In the cauda group one resection was done on a spastic and the other two on hypertrophied sphincters. There were four other patients with spastic sphincters that were not operated upon. In the civilian group only two patients needed and received resection. They were both patients that had had long periods of poor bladder therapy elsewhere before coming under my care and were both transected cord cases.

The only essential difference in these two series is found in the immediate and early method of dealing with the bladder. None of the patients treated from the start with tidal drainage and hence not subjected to prolonged periods of stretching, contraction or infection of this organ gave any evidence of any sphincteric condition that would necessitate resection. On the other hand, eleven patients whose bladders had not been similarly treated but had rather been subjected to a variety of physiologic insults did require resection of their internal sphincters before the organ could empty itself. To be sure, except for one patient who developed an incurable incontinence after the operation, all had a satisfactory postoperative result but this is certainly not enough to justify indiscriminate use of the procedure in "paraplegics" especially in the light of the success of the non-operative methods used in the larger series. Finally, in consideration of the incidence of the conditions calling for resection in the smaller series it is obvious that under similar conditions, if the operation has the widespread need that has been suggested, an analogous series three times as large should be expected to produce as many or more patients needing operation. The fact is that no resections were either indicated or necessary. The operation of resection *per se* will not be needed, therefore, to assure emptying of an "automatic," reflex or controlled bladder and if it is needed, is evidence of improper earlier therapy.

Flaccid Sphincters. Certain patients in both series had flaccid or completely atonic urethral sphincters. There were twelve such cases in the service and one in the civilian group. All occurred in patients with cauda equina injuries either alone eleven or in combination with an injury to the lumbosacral cord two. All showed evidence of denervation, both sensory and motor, of the peripheral field supplied by the sacral nerves. By cystometrogram four had atonic, eight autonomous and only one a reflex bladder. The conclusion is inescapable that the sphincters and all but one of the bladders had been denervated as the result of the central nervous system injury. As far as I know or can find out, this condition has never before been either described or recognized in connection with cauda equina or low spinal cord injuries. The constant wearing of a urinal is the only present method of dealing with the incontinence that goes with it.

Spasm. The third major impediment to the rehabilitation of patients paralyzed as the result of spinal cord and cauda equina injuries is "spasm." The most disabling variety of spasm is the involuntary variety, usually but not necessarily adductor-flexor in type, which involves not only the lower extremity but the lower back and abdominal muscles as well. In experimental animals, this is a constant accompaniment of an anatomic transection of the spinal cord at any level above the conus, and has been described under the term "mass reflex." It has been said not to develop in association with non-transecting cord injuries.[16] In any event it does not put in its appearance until spinal shock has worn off. In man the situation, although described as being the same as in animals, appears in the light of greater experience to be somewhat different. Involuntary spasm or the "mass reflex" is not, for example, a constant accompaniment of transection nor is its absence pathognomonic of a non-transecting injury. If all the transected and

partial cord injuries in the two groups are combined and only those patients are included that have lived three months or longer and thus have had an opportunity to develop spasms, there are ninety-one transections and 148 partial cord injuries to be considered. (Table VIII.) Of the transections—and that means loss of all sensory and voluntary motor function below the level of the cord injury—50 or 55 per cent had spasms. Of these only thirty-seven or 41 per cent had severe enough spasms to justify therapy. In the light of these figures it is impossible to contend any longer that involuntary spasms or the "mass reflex" is a necessary accompaniment of transection of the spinal cord in man. Of the 148 partial cord injuries seventeen or 11 per cent had spasms, fourteen or 9 per cent being severe enough to prevent ambulation completely. It is evident that invaliding involuntary spasms that are indistinguishable from those described as constituting a "mass reflex" *do* occur in patients who retain sensation and a varying degree of voluntary motion below the level of a partial cord injury.

These considerations are of great importance because the responsibility rests squarely on the surgeon's shoulders to determine by every means in his power the degree and type of injury that has been done to the spinal cord before committing himself and his patient to any such destructive operation as anterior rhizotomy in his attempts to relieve this disabling condition.[1] This is more true because this may be the only way to control the spasms and make rehabilitation possible. In patients with transected cords the usual root section can be justifiably carried out. If the cord lesion is known to be only a partial one, however, the rhizotomy must be done in such a way as to preserve the already existing voluntary motion. I believe that this important differentiation can be met only by actual operative visualization of the injured areas. Moreover, this should be supplemented by electrical stimulation of the cord above and below that area before a rhizotomy is justified. On that basis, and with the above requirements met in every case, only twenty-seven patients or 21 per cent have had rhizotomies out of this group of eighty transections and forty-eight partial lesions. Four additional service patients would not allow the operation to be performed and twelve were still to be done in October, 1947, on service patients that were still hospitalized. On the other hand, fifty-four exploratory laminectomies have been advised and fifty-three performed on the seventy-two discharged service patients in the transection and partial cord injury groups. Forty were done on the transected and thirteen on the partial cord injury

TABLE VIII
INCIDENCE OF INVOLUNTARY SPASM

	Died within 3 mo. of Injury			Total Cases			Cases That Could Have Developed Spasm			Severe Spasm			Mild Spasm			No Spasm			No Note		
	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total	Service	Civilian	Total
Transection of Cord	0	33	33	47	77	124	47	44	91	16	21	37	13	0	13	17	21	38	1	0	1
Partial Cord Injury	0	58	58	25	181	206	25	123	148	11	3	14	2	1	3	12	119	131	0	0	0
Cauda Equina Injury	0	0	0	28	44	72	0	0	0	0	0	0	0	0	0	28	44	72	0	0	0
Totals			91			402			239			51			16			241			2

cases. I am convinced that this realistic approach to the treatment of spasms has materially hastened and greatly improved the rehabilitation program and, at the same time, has avoided doing harm to and depriving any patient of any useful neuromuscular activity he would otherwise have had.

RESULTS OF TREATMENT

In measuring the success or failure of any program directed toward rehabilitating "paraplegics" there are many other factors to be considered beside the patient's ability to ambulate. Failure to itemize *all* of these factors casts doubt upon the claims of the sponsor.

Ambulation. It is not enough to know that a patient can get about; one must also know *how much* he can get about. We do not consider that a patient has satisfactorily finished his ambulation training until he not only can but prefers to wear his braces all day, is able to walk a thousand yards without stopping, to go up and down all stairs no matter whether they have a hand rail or not, to go up and down curbs freely, to get in and out of all types of chairs, to get himself off the ground without help if he falls, to walk in city traffic unaided and safely, to use all public conveyances without help, to live in his original home without demanding significant alterations, to use the toilet and bath-tub as usual and to live as normal a social and work life as the enforced use of braces and crutches will permit. We have met these self-imposed requirements in sixty-two or 62 per cent of the service patients that form the subject of this report. Our greatest success came in the group with cauda equina injuries. All but three of these twenty-eight patients or 98 per cent acquired all the skills listed above as well as some others. Twenty-three or 49 per cent of the patients with transected cords and fourteen or 56 per cent of those with partial cord injuries did the same thing. A less satisfactory but workable rehabilitation satisfied another group. They are able to walk only 500 yards, do not wear their braces as long, use only stairs with rails, have to be helped into public conveyances, and in general are less active and skilled. As a result certain alterations have had to be made in their homes. This and the preceding group were able to obtain and learn to drive an automobile. They usually were licensed on our recommendation by the state authorities. The combination of available transportation of their own, adequate social activity, plus an inability or disinclination to perceive the benefits that would accrue to them by finishing the full course of ambulatory training proved too much for seventeen such patients. They were discharged with our blessing but without our approval, the more so as there was no way by which they could be detained. It was hoped that perhaps, some of them would return later when they had learned by practical experience how much they were missing. Eleven came from the transection, four from the partial and two from the cauda equina group of injured. Thus seventy-nine or four-fifths of 100 "paraplegics" have been discharged during the past year able to ambulate enough to live at least a restricted social and work life.

Twenty-one did not attain ambulatory ability commensurate with the above. Two of these were discharged dead without ever getting out of bed and while still in the hospital, seven left in wheel-chairs and twelve were satisfied with a swing-through gait but no more. These last twelve were the only patients of the twenty-one who might have made something more of their opportunities but did not do so. The reasons follow: one still remains A.W.O.L., four were discharged for disciplinary reasons and seven left against medical advice.

All but one of the seven that were discharged in wheel-chairs and without having had any ambulation training were irreclaimable at the time they left. Three of them refused even to permit measurements to be taken for braces stating that they had no intention of ever learning to ambulate. One also had disabling

spasms which could be corrected only by surgery but would not permit this to be done. Two of the remaining three were limited to wheel-chairs by a floating femur with osteomyelitis of the head, neck and acetabulum, and by quadriplegia complicated by a paranoid schizophrenic personality, respectively. The remaining patient was transferred at his own request to another Veterans' Administration Hospital for his ambulation training.

As far as ambulation goes our program was a failure in twenty-one of the 100 patients. This was ascribable to the untreatable nature of the disease in five; to the patient's own peculiarities in nine and to the doctor's inability to hold the patient's interest in seven.

Control of the Bladder. As important to the patient from the point of view of a decent social and work life as ambulation is his ability to control his bladder and bowel. Because it can be attained if the patient cooperates and the doctor makes use of the knowledge available to him, I believe that anything short of perfection in these two functions is unjustifiable except where there is actual anatomic deficiency. Thus a patient is only considered to have obtained maximum benefit from the therapy he has received when he is able to go through a normally active day without getting wet and can regularly sleep through the night without having to get up and without getting wet. The wearing of a urinal or other apparatus is not countenanced unless the patient has loss of control by reason of irreversible anatomic changes. If his internal and external sphincters are atonic or his bladder has been allowed to stretch to a point where its tonicity is permanently destroyed or to shrink to a point where its destensibility is gone and its capacity reduced to only 2 or 3 ounces or less, then and only then is the wearing of some form of urinal permitted. The only exception that is made is for the patient who has to take a long trip on which he will be unable to empty his bladder at the regular intervals that he is accustomed to. Then a urinal is considered justifiable as insurance against wetting. In particular, we feel very strongly that an "automatic" bladder need not and should not be considered a satisfactory end point by either the doctor or the patient.

An analysis of the urination situation in the 100 discharged patients demonstrates that we have attained our self-imposed goal. Forty-five patients had acquired twenty-four-hour control by the time they left the hospital. Nineteen had day control only. They did not have night control because they would not adjust their water intake.[16] If they had been willing to do this, they could have had twenty-four-hour control. Forty-four of the sixty-four that had either twenty-four-hour or day-control used no urinal at any time. The other twenty as well as eight patients who had no control used a urinal on trips only. There was no information as to use of urinal by ten patients. Thus forty-five of ninety-eight, (46 per cent) had full control and sixty-four or 66 per cent had day control. Forty-four of ninety or 49 per cent did not use a urinal at all and twenty-eight or 31 per cent used a urinal on trips only.

Thirty-six patients were discharged without bladder control. Twenty-four or 27 per cent used at urinal at all times and four or 11 per cent used tidal drainage at home. In fifteen the lack of control was the patient's own fault. They usually preferred not to take the trouble to either learn bladder training[16] or to regulate their water intake and in consequence not only had no control, but also had to use a urinal constantly. In three the lack of control was inherent in the patient's condition (two deaths, one quadriplegic patient), in ten it was inherent in the bladder and in seven in the sphincters either alone or in conjunction with the bladder condition. The cause of this latter difficulty was the denervation of the organ and its musculature by the cord or cauda equina injury with the production of either atonic or autonomous bladders and flaccid sphincters. In only one patient was the lack of control unexplainable and hence charge-

able to the doctor. Thus it has been possible to teach all except one of the patients that were physically capable of learning to do so and that were cooperative and willing to take enough time and trouble, to have twenty-four-hour control of their bladders without the use of a urinal at any time except as a safeguard on long trips when the bladder could not be emptied at the required intervals.

Bowel Control. It is only slightly less important for these patients to be able to control their bowel. Here again we believe that we are derelict in our duty if anything short of twenty-four-hour control without leakage or soiling, the use of enemas, digital emptying or any similar procedure and with only minimum use of a bland cathartic is countenanced as a satisfactory end result. We were able to discharge seventy-one of ninety-three patients as having met these requirements. Information as to the bowel condition on discharge is not available in seven. Only six were taking enemas regularly at discharge.

Sixteen patients were discharged without bowel control. In nine this was the patients' fault in that they were discharged for disciplinary reasons two, were A.W.O.L. two or were uncooperative five. Six had no control because of their physical condition, one having died and five being afflicted with atonic sphincters because of denervation as the result of the cord or cauda equina injury. One other left to go to another Veterans' Administration hospital for completion of his treatment. Thus, there were no patients that lacked bowel control on account of the doctor's failure.

Bed Sores. As noted above, 71 per cent or seventy of the ninety-five discharged patients about whom the information is available, had bed sores that required treatment during their stay. In ten the bed sores were still present on discharge. Four were given medical discharges despite that fact. The decubiti were small and were located in the gluteal fold, on the sacrum, over the trochanter and on one heel, respectively. One patient died in the hospital, two were discharged for disciplinary reasons, two left against advice and one was transferred to another Veterans' Administration hospital at his request. The scars in nineteen others were adherent but they were either so placed or so small that it was not anticipated that they would break down. Twenty-six had non-adherent and fourteen padded scars. In fourteen there had been no note made about the condition of the scar. Thus, only four patients out of fifty-six that had had bed sores and about whom a note was made relative to their condition at discharge can be said to have been allowed to leave the hospital before a proper end result had been obtained.

Pain. Pain did not prove to be the major problem that was expected. Sixty-five of the ninety-nine patients about whom this information was available had no pain on discharge. Another twenty-seven had minor degrees of pain that were no more than bothersome. This type of pain was distributed about equally among the transected (ten), the partial cord injuries (nine), and the cauda equina cases (eight). Active treatment was neither indicated nor necessary in any of these cases. Six cases had major disabling pain on discharge. All were in the cauda equina group of injuries. One patient in this last group died from the infection which caused his pain. Another complained of severe pain in his rectum the cause of which was unknown and which was not relieved at the time of his discharge against medical advice. The available records gave no information about his treatment. Of the remaining four, three had a radicular type of pain. Two of the three had posterior rhizotomies and one a cordotomy in addition without relief. The fourth left without having received any active therapy and after having completed his ambulation training, but with an autonomous bladder, spastic urethral sphincters and no urinary control. His bowel control was satisfactory. This last case had severe leg pain of an atypical type. He was a constitutional psychopath, would not eat properly and was frequently under the

influence of alcohol. He refused all treatment except drug therapy with opiates and barbiturates and was discharged against medical advice.

Spasms. Spasms may be enough to prevent rehabilitation even though all other disabling conditions have been cared for. It is therefore essential to know about their presence in the discharged patients. Such data are available in ninety-eight patients. Sixty-two were discharged without spasms and twenty-two with minimal spasms that caused no interference with ambulation or bladder and bowel control. Fourteen had spasms that were bothersome or worse at discharge. Ten of these had had a transection and four a partial injury of the cord. Only three cauda equina cases had spasm of any type. It was minimal and in every instance there was an associated injury to the lower end of the cord in addition to the damage to the cauda equina.

Three of the fourteen patients with severe spasms were disabled by them. One of these refused an anterior dorsolumbar rhizotomy and became an able ambulator nevertheless; one had an ineffective anterior rhizotomy and was discharged against medical advice while still in a wheel-chair and the other was a schizophrenic quadriplegic who was discharged in the same way and in the same condition. In nine the spasms were only bothersome. Two of these were discharged for disciplinary reasons—one after refusing rhizotomy—but not on that account; four were discharged against medical advice one having refused anterior rhizotomy. None of these six were able to ambulate beyond the first stages at the time of their discharge. One was sent to Cushing Veterans' Administration Hospital having just graduated from a wheel-chair existence. The other two were able to ambulate fully. They were given medical discharges despite the presence of spasm. One patient had minimal spasms. He refused an anterior rhizotomy and was given a disciplinary discharge for other reasons. The last was given a disciplinary discharge while still in a wheel-chair, the degree of his spasms at that time not having been noted. Thus the presence of significant spasms at discharge may be considered the patients' fault in eleven and the doctor's fault in two. In connection with this it should be noted that only thirteen rhizotomies were performed and only seventeen recommended in this group of 100 patients, the other four having been refused by the patients. Of the thirteen that were carried out, ten were anterior dorsolumbar and three posterior in type. One patient was relieved of disabling typical abdominal flexor spasms associated with a mid-thoracic transection of the cord by injection with novocaine of the appropriate peripheral nerves at their exits from the intervertebral foramina.

Sexual Status. It is not possible to give any figures on the sexual status of the patients in this series. An investigation started one year ago in an endeavor to bring some order out of the chaos of irresponsible statements that are in the medical literature has demonstrated that the problem is so complex that much more work and time must be spent on it before any conclusions that even approach reliability can be drawn.

Patient's Attitude toward the Treatment He Has Received. In addition to the physical one this group of injured has a tremendous psychologic problem to face. We believe that we can to some degree, measure the success of our treatment of the latter by delving into the psychologic field far enough to attempt to determine the patient's own reaction to this therapy. These patients, therfore, were all asked at discharge whether or not they had been satisfied with the treatment they received while under the Veterans' Administration care at Cushing General Hospital. Answers were obtained in ninety-eight of the 100. Seventy-nine expressed themselves as satisfied, eight as not satisfied and five asked to be transferred to other Veterans' Administration Hospitals. Five were given their discharges for disciplinary reasons, fourteen

left against medical advice, three were A.W.O.L. and two died in the hospital.

Of the eight that left unsatisfied with the treatment, six had transected cords and two partial cord injuries. One of the transected patients left after eight operations which all originated with the implantation by some surgeon overseas of a Lane's plate in a dirty spinal wound. Four were obviously emotionally upset, one having his judgment warped by his poor general condition, another because attempts were made to force him to ambulate against his will, a third because "everything was wrong" and the fourth as the termination of a consistently and continuously recalcitrant attitude. The sixth left without giving a reason. The first of the two partial cases was a schizophrenic quadriplegic and the second left following and probably because of an unsuccessful anterior rhizotomy.

Discipline has not presented a major problem. I consider that this is traceable to two reasons: First, all patients are given a schedule which keeps them occupied in some activity looking toward their more rapid discharge and more complete rehabilitation and, second, because the attitude of the staff is that the patients are in the hospital only temporarily and as a means to an end. Repeated infractions of the work schedule, excessive use of alcohol, foul or obscene language and major insubordination are followed by a hearing of all parties before a board and the discharge of the patient for disciplinary reasons if the charges are approved by the board. This procedure is adopted as a last resort only. The local paraplegic association has proved extremely helpful in maintaining discipline once its members understood that cooperation with the medical program meant quicker and more complete rehabilitation. Only five patients were discharged for disciplinary reasons during the year. Two were because of the persistent use of alcohol; one because of being persistently A.W.O.L.; one because he refused to take any part in the medical program and the last because of driving an automobile on the public highways before he was able to do so, without our permission and without being licensed by the state.

Training for and Provision of Employment after Discharge from the Hospital. The most difficult, the most important and probably the least well handled of the whole problem of rehabilitating the spinal paralytic is to provide him while in the hospital not only with the potential ability to earn his own living but with employment after he leaves the hospital. No matter how medically fit these patients are on discharge, if the mechanism is not provided for them whereby they can earn their own living in their community and thus maintain their self-respect after leaving the hospital, all the professional care that has been lavished on them will be wasted. It is unreasonable to expect the average family or friend of a "paraplegic" to assume the same detached attitude toward him that they would toward a normally equipped wage-earner. They cannot and will not make him work even if he is able to do so and will have no part in forcing him to train himself for work if he has not already acquired that ability. Indeed, the weight of community public opinion as well as the emotional response of his family will make it extremely difficult if not impossible for the "paraplegic" who wants to work in spite of his handicap to do so. His family feels guilty if they do not see that he need carry no responsibility in the home. The community through the various charitable organizations takes the same attitude. Employers, in the main, regard the employment of a "paraplegic" not in the light of buying competent labor but rather as a noble gesture or as "part of their war effort" and lacerate the "paraplegic's" pride and self-respect by paying little or no attention to his skill or his worth as a member of their organization. Only prolonged education of the employing and employable population and of the parents and friends of these patients can correct this. Rather than force or allow the "paraplegic" to combat this attitude unprepared

he must be given the opportunity to learn a trade that is within his physical capabilities while in the hospital, and gainful employment in that trade must be waiting for him on his discharge. We have failed badly in meeting these responsibilities. The reasons are many and complicated but the reason that most people will assume as the chief one is not a major factor. This is the unwillingness of these patients to work. Only six out of the 100 that were discharged made the statement that "they did not intend to work but were going to let the government take care of them" for the rest of their lives. This is certainly no more than the number of employables with that attitude in the community at large. It is only surprising that it is not larger. These patients, being totally disabled, will get paid at least $360 a month by the Federal government and in certain states will receive additional sums that bring the figure close to $500 a month for the rest of their lives. This is sufficient for almost anyone to live on comfortably without further income and it is a most effective deterrant to starting on a new, strange job. On the other hand, it is less than these men deserve from the country and any effort looking toward its reduction is unthinkable. We should, however, see to it that they are in a position to use this money to their own best advantage. This means gainful labor in the case of a male adult.

We have information on fifty-five potential wage earners relative to their capacity and willingness to do gainful labor on discharge from the hospital. Thirty of this group had a job at gainful labor or its equivalent in educational training. Twenty-five did not. The forty-five other patients fell into a different category. It was physically impossible for three to hold a job on discharge. Seventeen separated themselves from the hospital by their own actions before arrangements for their training and placement could be completed; two being A.W.O.L. constantly, seven having left against medical advice, five having been discharged for disciplinary reasons and three having gone to other Veterans' Administration hospitals. There was no information about employability or employment in the records of twenty-five patients.

It seems to me that despite the quality of medical care, which the figures show to have been good, and in spite of the fact that unwillingness on the part of the veteran to work played no part in the picture, we have fallen far short of the necessary requirements for rehabilitating these patients. Our shortcoming lies in failing to see to it that they are trained in wage-earning skills during their hospital stay and that they are provided with gainful labor in accordance with this training on their discharge to civilian life again.

SUMMARY AND CONCLUSIONS

1. The results of the treatment of 212 veterans suffering from wounds and injuries of the spinal cord and cauda equina —including "paraplegics"—at Cushing Veterans Administration General Hospital during the year from October 1, 1946 to October 1, 1947, is reported.

2. One hundred patients were discharged and 112 remained for further treatment. There were forty-three readmissions.

3. Seventy-nine patients were fully ambulatory at the time of discharge.

4. Of the twenty-one that were not ambulatory at discharge five were failures because of the untreatable nature of their disease, nine because of the patients' own peculiarities and seven because the doctor failed to hold their interest.

5. Sixty-four of the 100 patients were capable of acquiring full control of urination: forty-five had twenty-four-hour control, nineteen had day control only, forty-four used no urinal at any time and twenty-eight used a urinal on trips only.

6. Thirty-six patients were discharged with no control of urination. In fifteen the lack of control was the patients' own fault, in three it was inherent in the patients' condition, in ten it was inherent in the condition of the bladder or sphincters or both and in one was unexplainable.

7. Seventy-one patients were discharged with full twenty-four-hour bowel control.

8. Pain was not a major problem. Only six patients (all cauda equina injuries) had enough pain at discharge to warrant treatment.

9. Disabling spasms were present in fourteen patients at discharge. Sixty-two patients were discharged without spasm and twenty-two with minimal spasms that caused no interference with the patients' ambulation or bodily functions.

10. All spasms when present were associated with a cord and never with a cauda equina injury.

11. Thirteen rhizotomies were performed, ten anterior dorsolumbar and three posterior in type.

12. Seventy-one patients had bed sores that required treatment during their stay. Bed sores were still present in ten at discharge.

13. Discipline was not a problem, only five of the patients having been discharged for disciplinary reasons.

14. Much still needs to be done in educating the public, these patients and their families as to the necessity of training these paralyzed veterans for gainful labor during their hospital stay and providing them with gainful employment and the ability to be self-supporting when they leave the hospital.

15. Evidence is presented by means of appropriate comparison with a civilian series of 313 patients with spinal cord and cauda equina injuries to demonstrate that: (1) The use of plaster of paris body casts is conducive to the formation of bed sores; (2) renal and bladder stones can be virtually completely prevented by the early persistent and proper use of tidal drainage alone; (3) need for transurethral resection of the internal urethral sphincter to restore micturition is evidence of improper earlier care of the bladder and is not otherwise obligatory in these patients and (4) the spasms characterized by the term "mass reflex" occur in both complete transections and partial injuries to the spinal cord in man.

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ACUTE ABDOMINAL EMERGENCIES IN PARAPLEGICS*

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DURING the past five years medical literature has stressed, time and time again, the fact that the post-war years would bring diagnostic problems to the civilian physician which he had not previously encountered.[1,2] The profession at large has been cautioned, even in apparently simple diagnostic problems, to bear in mind the possibility of recurrent malaria and various other exotic diseases when dealing with returned overseas veterans. In addition to the tropical diseases to which the returned military may be heir, there are other formerly rare maladies which the war has made common place. It was estimated that at the close of hostilities there were approximately 2,000 paraplegic patients in army, navy and veteran administration hospitals.[3] Because of the progress made during the war years in the management of these patients, the majority will be rehabilitated to the point of assuming a happy and useful life in the civil community. As this group of paraplegics returns to civil life they will bring to the civilian physician many diagnostic and therapeutic challenges.

One of the most difficult problems which the paraplegic patient presents is that of the acute abdominal emergency. Because of the extreme debilitated state of such patients when returned from overseas areas of combat, because of the foci of infection in the decubitus ulcers and genitourinary tract, because of the tendency of these patients to genitourinary calculus formation and because of their low resistance to intercurrent disease, acute abdominal emergencies were seen far more frequently in a group of patients in a paraplegic center than in an equal number of healthy young adults. It is believed that these patients after leaving these centers will continue to present perplexing syndromes of abdominal distress to the civil physician. The presence of a profound neurologic lesion, which usually results in anesthesia below the level of injury, makes the evaluation of these episodes an extremely difficult matter. The purpose of this paper is to enumerate certain factors which may be of assistance in evaluating symptoms of abdominal distress in the paraplegic patient.

MECHANISM OF VISCERAL PAIN CONDUCTION IN COMPLETE SPINAL CORD TRANSECTION

It has been definitely established that the abdominal viscera are supplied with sensory nerve endings, and that painful sensation may be conducted from the viscera to the spinal cord by way of the thoracic, splanchnic, hypogastric and pelvic nerves and thence over the posterior nerve roots.[4] White has pointed out that even though these visceral sensory neurones resemble sympathetic motor neurones, they are anatomically as well as physiologically different. He states further that it is incorrect to refer to sympathetic pain fibers, but best to think of the visceral innervation as composed of mixed nerves, containing a large preponderance of sympathetic motor fibers with a small admixture of sensory fibers which belong to the posterior spinal root.[5] Nevertheless, despite the fact that visceral sensation is conducted by the posterior root system in a similar manner to the pain conduction from the abdominal wall or from the extremities, there must be a dissimilarity between the pathways of reception of

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visceral sensory impulses and somatic sensory impulses. Many paraplegics with a physiologically complete transection of the spinal cord cannot perceive any sensory stimulation of the somatic type below the level of spinal cord transection, but can perceive painful stimulation of the viscera whose sensory fibers are believed to enter posterior roots below the level of transection or compression of the cord.

We have seen several paraplegic patients with abdominal pain who were known, by means of exploratory laminectomy, to have complete spinal cord transection, but who, nevertheless, developed abdominal pain from disease of viscera whose sensory nerve fibers are reputed to enter roots below the level of transection. Livingston states that it is particularly notable that visceral pain which is satisfactorily relieved by bilateral chordotomy often gradually returns, and that visceral pain pathways may have accessory pathways by which they reach the sensorium.[6] Karpus and Kreidl suggest that such accessory pathways may be represented by relays of short neurons close to the spinal gray matter,[7] and the escape of such pathways from damage might account for the presence of visceral sensation in the absence of all somatic sensory perception in a rare case. Foerster has suggested that these visceral impulses of pain may pass upward for several segments in the sympathetic ganglionic chain before entering the cord.[8] This is believed to be the explanation in several of our own cases.

One case cited below, however, cannot be satisfactorily explained by any of the above postulations. This young male was rendered quadriplegic following fracture of the fifth cervical vertebra. Exploratory laminectomy revealed a complete transection of the spinal cord in the region of the sixth cervical nerve root. Nevertheless, this patient developed sharp stabbing pain in the epigastrium due to a gastric ulcer, the presence of which was confirmed by x-ray. In this case it is impossible to assume that the afferent pain impulses were transmitted cephalad either by accessory pathways in the cord or by fibers running in the sympathetic chain to enter the cord above the site of the transection. The vagus nerve has not yet been demonstrated to carry painful impulses *per se* from the abdominal viscera.[9,10] White states that while the vagi carry some afferent reflex stimuli and a part of the sensation of nausea, they are not known to carry any definite pain sensation.[4] However, in a personal communication White states that he has recently found good evidence that vagi can transmit cardiac pain referred to the jaws after all the sympathetic fibers are cut. Chapman has found that pain caused by distending balloons in the duodenum and jejunum is relieved by splanchnicectomy. However, when the balloon is in the esophagus, neither total sympathectomy nor spinal anesthesia as high as the first thoracic segment stops the pain.[11] His conclusion is that this midline, diffuse, aching sensation must be transmitted over the vagi. The recent work on vagotomies in peptic ulcer has led to much speculation on the mechanism of pain relief in these cases. Alvarez, in accord with most other investigators, states that the sensory pathways are not interrupted by this operation.[9] Livingston suggests that impulses, finding themselves blocked from their customary pathways, eventually find new or previously unused pathways for reaching the sensorium.[12] Therefore, because no other pathway of pain conduction can be postulated in the case cited, and in view of the findings of White that some cardiac pain can be transmitted via the vagus, and of Chapman that some esophageal pain can be transmitted in the same way, we believe that in at least in certain instances painful impulses from the stomach may be transmitted via the vagus nerves.

Differential Diagnosis. It is beyond the scope of this communication to present the entire differential diagnosis in cases of acute abdominal crises. Certain it is that

all those acute abdominal maladies which may afflict a normal healthy young adult may also occur in a paraplegic subject. However, there are certain of these conditions to which a patient with a cord lesion is particularly liable, and it is to these entities that we should like to direct attention in this report.

In all but one of the cases presented below, comprising both complete and incomplete spinal cord transections at varying levels, abdominal pain was one of the presenting symptoms of the episode referred to as an acute abdominal crisis. In each of these selected cases, neurologic examination revealed the appreciation of touch and pain stimuli to be absent below the level of the spinal cord lesion. The site of the lesion varied in these cases from the sixth cervical to the eighth thoracic spinal segment. Nevertheless, in all but one case, abdominal pain was a presenting symptom. In practically every case the pain was described by the patient as a dull oppressive pain which was generalized or unlocalizable. In one case the pain was sharp and stabbing. In each instance, with the exception of the case referred to, the patient complained of terrible pain somewhere in the abdomen and the pain often reached excrutiating proportions but did not become localizable nor sharp. Nausea and vomiting were present in all cases, occasionally preceding onset of this unusual type of pain, and sometimes becoming so severe as to be considered the major symptom. It is our impression that paraplegic patients are more prone to become nauseated and vomit from various and sundry conditions than are non-paraplegics.

Physical signs are usually masked, inasmuch as abdominal rigidity may be absent or unrecognizable due to muscular flaccidity or spasticity, dependent upon the neurologic lesion. Abbott recently presented a report on the importance of recognizing abdominal rigidity which is often present in patients who are paraplegic due to concussion of the spinal cord.[13] He points out the fact that this rigidity of the abdominal musculature is often indistinguishable from that due to intra-abdominal pathologic processes. Tenderness was present in only one of the cases presented and is almost universally absent in these patients. Slight elevation of temperature was present in all of those cases and has been found to be of differential diagnostic value only when it suddenly spikes to 103°F. or above, in which case it has almost always been found to be due to an exacertation of the ever present urinary tract infection. The white blood count was markedly elevated in every case herein presented, and was of value only when we had previously established a "normal" base line for the patient in question. The absence of borborygmus has proved to be a particularly valuable sign when it has previously been present, thus there may be a perversion of the signs and symptoms.

When a paraplegic patient complains of pain, the neurologic lesion must first be ruled out as the producing cause. Pain resulting from a spinal cord lesion is usually radicular in distribution, and more often is sharp and knife-like in contrast to the dull, oppressive, generalized pain of which these patients complain in case of visceral disease.

The next most common cause of abdominal pain in a paraplegic, usually accompanied by nausea and vomiting, is an affection of the genitourinary tract. These patients, in practically every case, have, at some time during their course, an indwelling catheter of the urethral or suprapubic type, and in almost 100 per cent of the cases there is at least a latent genitourinary tract infection. Episodes of pyelitis are usually accompanied by severe unlocalized pain of the type already described. However, a sudden elevation of temperature to 103°F. or above usually points to the diagnosis in these cases. Inasmuch as the urine of these patients is usually loaded with white cells and bacteria, urinalysis is generally of no

diagnostic value. However, if frequent routine urinalyses are obtained, comparison with these at the time of the acute episode may reveal a change of diagnostic value. These episodes will usually respond within a few hours to chemotherapy.

An equally frequent cause of abdominal pain, accompanied by nausea and vomiting, in this group of patients is urinary tract calculus. It is well known that the atrophy of disuse leads to the mobilization of calcium with increase in the calcium content of the urine, but it must be mentioned that disuse is not alone responsible for the tissue breakdown in paraplegics. The regularity with which mild febrile episodes or operative procedures precipitate disturbances of tissue metabolism, as evidenced by loss of previously gained weight with negative nitrogen balance, breakdown of healed decubitus ulcer and rapid formation of renal calculi, suggests a disturbance of hormonal origin. While specific data are lacking in our cases, it is our impression that both anterior pituitary extract and testerone have a beneficial effect in arresting these bouts of katabolic excess.[14] Be that as it may, paraplegics are particularly prone to develop renal calculi. The pain in these cases is severe, oppressive and cannot be localized, although in the case of renal calculus presented in this report, percussion over the affected kidney caused increase in the severity of this peculiar type of pain. The temperature is usually elevated to 99° to 100°F. The diagnosis depends on roentgenologic demonstration of the calculus. In every case of suspected abdominal distress in a paraplegic patient, a flat plate of the abdomen and an emergency intravenous pyelogram are indicated. Here again, previous routine roentgenograms for comparison are of obvious value. The possibility of a perforation of the urinary bladder must be constantly borne in mind because of the friable condition of the infected bladder which develops during the course of this disease.

When the examiner is convinced that the neurologic lesion and the genitourinary tract are exonerated as the cause of the acute condition, all other causes of acute abdominal emergency must be ruled out, bearing in mind the peculiarities in the physical findings which have already been discussed. Pain may sometimes be absent in spite of an acute visceral disease. The examiner must also be aware of the patient's psychologic status. We have noted that realization of and adjustment to the paraplegic state occurs in step-like fashion; some of the steps are big ones and are frequently associated with more or less objective psychosomatic manifestations.

CASE REPORTS

Case I. V. R., a nineteen year old white male, was rendered quadriplegic, with complete loss of motor power and all sensory modalities below the sixth cervical dermatome, on September 9, 1945. A laminectomy on March 1, 1946, revealed a complete transection of the spinal cord at the level of the fifth cervical lamina. In April, 1946, the patient began to complain of intermittent episodes of sharp stabbing pain in the mid-epigastrium. Physical examination was negative except for abdominal rigidity which had, however, been present since admission to the hospital. The pain was most marked at night and was relieved by ingestion of milk. The episodes of pain became more frequent and increasingly severe, and were occasionally accompanied by vomiting. A contrast medium gastrointestinal roentgenogram revealed the presence of a gastric ulcer. The symptoms were completely relieved by institution of an appropriate dietary regimen.

Case II. H. K., a nineteen year old white male, received multiple gunshot wounds of the back on March 2, 1945, and was immediately rendered paraplegic, with complete loss of motor power and all sensory modalities below the eighth cervical dermatome. From the time of his hospital admission he was severely depressed mentally and had frequent episodes of nausea and vomiting, lasting about twenty-four hours. Routine studies were negative, including intravenous pyelograms, and it was believed that these episodes were psychogenic. Both the general surgical and psychiatric consultants concurred in this opinion. On March 17, 1946,

one year after injury, the patient became extremely nauseated and vomited several times. The vomiting persisted and on the second day became more severe and the patient complained of dull oppressive pain "somewhere in the abdomen." Physical examination revealed the musculature of the abdomen to be flaccid, but percussion over the right costovertebral angle increased the vague pain. The white blood count was not significantly altered from this patient's average count, and the urinalysis revealed many white cells with few red cells, but these had been present previously. An intravenous pyelogram demonstrated several calculi in the left renal pelvis and ureter with blockage of the ureter. The symptoms subsided following operative removal of these calculi.

Case III. W. C., a twenty-five year old white male, was wounded by machine gun fire on February 24, 1945. He suffered complete loss of motor power and all sensory modalities below the seventh thoracic dermatome. He complained constantly of lancinating pain in the right upper abdominal quadrant which was not relieved by analgesic medication. On August 27, 1945, a laminectomy revealed a shrunken, non-viable spinal cord at and below the eighth thoracic segment. A bilateral radical section of the spinothalamic tracts was performed to relieve the pain of which the patient constantly complained. On November 21, 1946, this patient's abdomen became distended and the patient complained of constant dull oppressive unlocalizable abdominal pain which became increasingly severe. Peristalsis was observed to become more prominent. Enemas brought about no relief and no fecal impaction could be demonstrated. The patient did not vomit until forty-eight hours after onset. Urinalysis was unchanged and the white blood count rose to 16,000 whereas it had previously maintained itself at about 11,000. An x-ray of the abdomen revealed a marked degree of dilatation of small intestinal loops. A typical stepladder effect associated with intestinal obstruction was present. Three days after onset an exploratory laparotomy was performed and revealed many adhesions of the bowel to itself and two main adhesive obstructions, one near the root of the mesentery and one attached to the bladder. These were released and symptoms subsided postoperatively.

Case IV. W. K., a twenty-five year old white male, became paraplegic as a result of an osteochondroma of the body of the eighth thoracic vertebra. Neurologically this patient demonstrated a complete loss of all sensory modalities below the eighth thoracic dermatome and spastic paraplegia in extreme flexion with mass reflexes. This patient complained bitterly of sharp knife-like pain throughout the lower abdomen and lower extremities. Because of the extreme flexion spasm and the constant pain, an intrathecal injection of alcohol was given, with immediate relief from both of these distressing symptoms. Any complaints of pain thereafter were adequately relieved by placeboes.

A month later the patient complained of pain in the lower abdomen which was relieved by sitting up in bed. He became extremely nauseated and vomited several times. The following day the pain was more severe and was sharp and stabbing in nature. Despite the fact that there was no somatic sensory perception below the eighth thoracic dermatome, superficial palpation over the suprapubic area resulted in exquisite pain. The diagnosis of perforation of the urinary bladder was made and confirmed at operation.

Case V. L. L., a twenty-two year old white male, was wounded by a shell fragment on February 22, 1945, which resulted in a fracture of the eighth thoracic vertebra and complete motor and sensory loss below the eighth thoracic dermatome. He was evacuated to the United States, and by the time of his arrival at this hospital, six months after original injury, he had developed a large sacral decubitus ulcer which was subsequently closed by a plastic surgical procedure. From the time of arrival the patient complained bitterly of sharp stabbing pain in the right upper abdominal quadrant. This pain was constant but was more severe at some times than at others. The pain was not of the radicular type and it was not believed that it was due to the spinal cord lesion. An intravenous pyelogram on January 31, 1946, revealed the presence of a large staghorn calculus in the right kidney, and it was believed that this might be the cause of the patient's pain. On February 6, 1946, a pyelotomy was performed. The postoperative course was uneventful, but the pain continued unchanged in character. On March 13, 1946, a laminectomy was performed and the cord was found to be completely atrophic

below the ninth thoracic segment. The posterior roots of T-9 and T-10 were sectioned, despite the fact that the cord appeared atrophic and non-viable at this level. The subsequent course of this patient was marked by complete absence of the pain of which he had previously complained.

Case vi. W. R., a twenty-two year old white male, was rendered quadriplegic following an automobile accident on September 9, 1946, in which the patient incurred a fracture of the body of the sixth cervical vertebra. There was a complete loss of all motor and sensory function below the level of the seventh cervical segment. On November 20, 1946, the patient became nauseated and vomited several times, being unable to retain any food or fluid taken orally. His abdomen became distended and the peristaltic sounds were exaggerated. The patient had no pain. All laboratory studies were uninformative as were x-rays of the abdomen and intravenous pyelograms. The only positive finding on physical examination was the presence of a hard fecal mass noted on rectal examination. Review of the nurses' notes revealed the fact that enemas had resulted in very little fecal evacuation during the previous ten days. All symptoms subsided following manual removal of the impacted feces.

SUMMARY AND CONCLUSIONS

1. The mechanism of visceral pain transmission is discussed and it is suggested that the vagus nerve may carry painful impulses from the stomach, and cases are presented to show that present ideas of possible pathways for pain transmission are inadequate.

2. Causes other than neurologic must be considered when a paraplegic patient complains of abdominal pain.

3. The importance and value of establishing a base line of physical findings and average laboratory data of paraplegic patients is stressed. Routine periodic check-ups are essential.

4. When a definite cause of abdominal pain in these patients cannot be established, surgical exploration is justified.

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EXOPHTHALMOS*

SOME PRINCIPLES OF SURGICAL MANAGEMENT FROM THE NEUROSURGICAL ASPECT

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AS an appendage of the nervous system, the eye may present indications of highly important and significant changes in intracranial physiology. Many and varied alterations occur in the optic nerves, retinae and vessels, and the diagnosis of hemorrhages, certain neoplasms and infections may be made ophthalmoscopically. Pupillary changes and extraocular faults in innervation may permit localization and even pathologic diagnosis of disease in the central nervous system. No neurologic investigation is complete without perimetric studies. With so many and such varied evidences of neuro-ophthalmologic disturbances, it would have seemed logical if the ophthalmologists had extended their interests to the treatment of the underlying intracranial conditions and had become the neurologic surgeons of today. Evidently, the traditions of this venerable specialty and the character of training of the eye specialist were determining factors.

At any rate, the general surgeon, prompted by the neurologist, undertook the treatment of intracranial disorders. In such conditions as presented themselves, the immediate and urgent need for information on the condition of the optic discs made it necessary for the surgeon to acquire some familiarity with ophthalmoscopy and perimetry. From this beginning his interest in neuro-ophthalmology grew and of recent years a mutual interest in the treatment of exophthalmos has brought the ophthalmologist and the neurologic surgeon still closer. Ophthalmologists have made notable contributions to the neurologic aspects of perimetry. These have come largely from their intimate association with neurologists and surgeons.

In more recent years articles on the diagnosis, pathology and therapy of conditions producing protrusion of the eye have been contributed by workers from each of the specialties as well as by those from both fields working in conjunction. Many orbital lesions can best be approached from the front. This applies to those occupying the anterior third or perhaps the anterior half of the orbit and more particularly to those placed laterally. Here, a considerable space for exposure is available whereas on the nasal side of the muscle cone it is quite restricted. The principal contributions dealing with the varieties and pathogenesis of primary intra-orbital growths and the surgical methods for removal of them have come from the ophthalmologists.

Operative procedures for the surgical treatment of lesions affecting the optic pathways, chiasm, the optic nerve and the region of the sphenoidal fissure have been developed by neurologic surgeons. The intracranial approach to lesions of the parapituitary region has become commonplace in neurosurgical clinics and is similar to that needed for the orbit. Although orbital surgery includes many procedures that can be performed through the comparatively clean, but nevertheless contaminated, approach between the lids, when access to deeper structures by an intracranial route through a clean field is

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required the task has fallen into the hands of the brain surgeon.

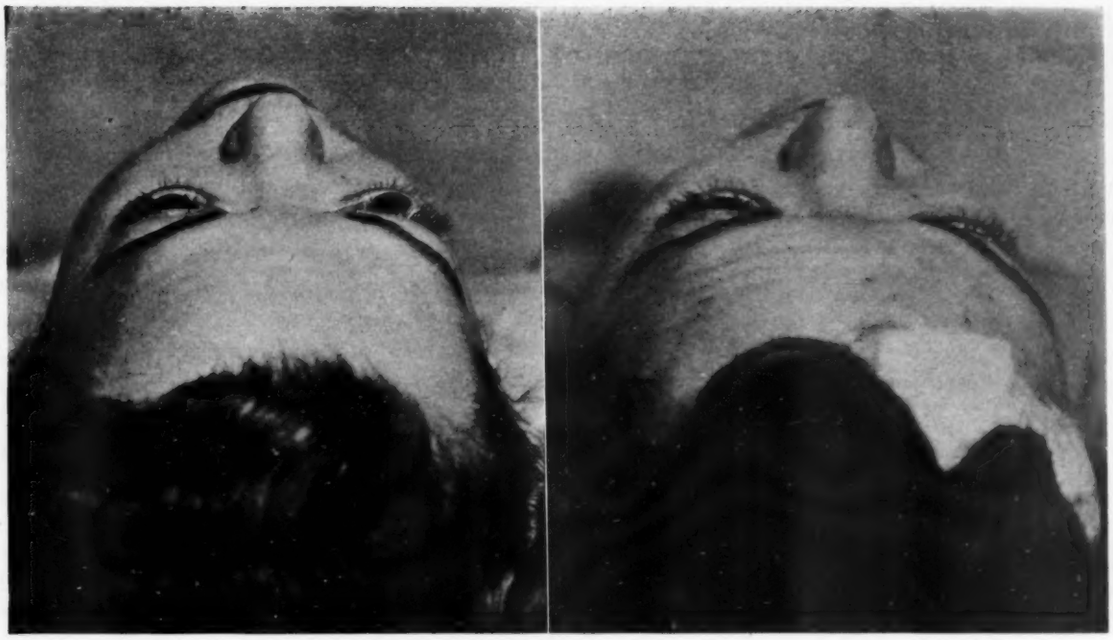
An increasing number of publications dealing with orbital surgery have appeared from both ophthalmologic and neurosurgical clinics. The field is still a fertile one. Much remains to be accomplished and major surgery of the orbit is not widely practiced. The treatment of certain malignant or eventually potentially irremovable growths in and about the orbit is lacking in a systematic approach. The Krönlein procedure and other restricted external operations, sympathectomy and x-ray treatment, which have comprised much of the therapy, are being replaced by better planned and more thorough treatment. Orbital conditions require the attention of the ophthalmologist, and study by him in conjunction with the pathologist, radiologist and neurologic surgeon should yield valuable dividends in improved treatment.

Under the heading of orbital tumors and unilateral exophthalmos excellent articles with liberal bibliographies have come from ophthalmologists, including Benedict, Reese, Davis and Spaeth, and from neurological surgeons, notably Dandy, Davis and Martin, Love and Poppen. With such recent exceptions as those mentioned, the neurologic surgeons have concerned themselves principally with the primary intracranial lesions in which protrusion of the eye has been one manifestation. The meningiomas arising from the neighborhood of the sphenoidal ridge and fissure, both extradural and intradural, the orbitoethmoidal osteomas of the nasal accessory sinuses and vascular lesions are among the more frequent conditions and a large literature has grown up in consequence.

The incidence of various pathologic processes is significant as a determining factor in the direction of the surgical approach to them. Benedict has estimated that intracranial involvement is present in 25 per cent of orbital tumors. Reese reports that in a large series of patients with unilateral exophthalmos the primary lesion in 63 per cent was located in the orbit. Dandy, reporting from the standpoint of a neurologic surgeon, stated that combined orbital and intracranial tumors were present in 80 per cent of his patients. Such figures make apparent the need for the combined efforts of the ophthalmologic and neurologic surgeon in developing this comparatively untilled field.

The problem in unilateral exophthalmos is one of diagnosis and location of the lesion to determine whether it is solely intraorbital, intracranial or both. The treatment is directed toward relieving the exophthalmos by removing the cause if possible. In bilateral exophthalmos the lesion is not a removable one and decompression is required. It is frequently difficult to determine whether an exophthalmos is unilateral or bilateral. Since the normal range of exophthalmometer readings varies from 11 to 20 mm. or greater, it is not always possible to state whether or not there is abnormal protrusion and exophthalmos for that particular individual. The literature abounds in reports in which statements and judgments of unilateral exophthalmos are based on appearances instead of measurements with an exophthalmometer. Such reports are of no particular value. Because of the normal range in the position of the eyes in the orbit and the misleading appearances produced by a stare due to variation in the interpalpebral space, even experienced examiners are easily misled. Without measurement one can usually determine a difference in the prominence of the two globes by looking down on the corneas from above and comparing their positions relative to the superior orbital margin. (Fig. 1.)

The determination of whether exophthalmos is actually unilateral or only apparently unilateral but in reality bilateral is of utmost importance in diagnosis. Unilateral protrusion is due to tumor. Bilateral protrusion has a more general cause. An experienced examiner may determine by palpation whether retrobulbar resistance is normal or increased in the suspected eye and so obtain a clue as to whether the eye



A B

Fig. 1. A, showing relative prominence of the globes as seen from above. B, note recession of the right globe following unilateral orbital decompression.

is pushed forward or not. The response of the globe to changes in intraorbital pressure has been likened to that of a tambour.

Usually, a difference of 5 or 6 mm. between the two eyes as measured with the exophthalmometer is seen with unilateral tumors but not from other causes. In diagnosis of masses in the orbit, x-ray has come to be of increasing value, both in plain x-ray films and by angiography. Roentgenographic study should include skull and bilateral orbital views. Deformity of the orbit, alteration or loss of bone density, enlargement of the optic foramen or sphenoidal fissure and changes in the sphenoidal ridge may be seen. Commonly, the epidermoids, meningiomas and osteomas produce characteristic pictures. On this subject the writings of Pfeiffer repay careful reading. With such x-ray studies and in the absence of neurologic findings indicating an intracranial involvement, the restriction of the lesion to the intra-orbital space may be estimated. The character of the ophthalmoscopic findings and perimetric fields, the displacement of the globe, the palsies and palpation give clues to the position of the mass in the orbit and the structures involved.

Ophthalmologic literature indicates the considerable frequency of intra-orbital inflammatory disease, often secondary to infected paranasal sinuses. Reese analyzed 174 consecutive cases of unilateral exophthalmos caused by tumors and tumor-like lesions. He excluded non-surgical exophthalmos associated with goiter, arteriovenous aneurysm, sinus mucocele, cavernous sinus thrombosis, oxycephaly and aneurysm of the ophthalmic artery. Of 109 lesions which were primary in the orbit there were: twenty-five hemangiomas, eighteen pseudotumors (chronic cellulitis or lymphogranuloma), thirteen meningiomas (these were not all primary in the orbit), ten dermoids and epidermoids, ten varieties of sarcoma, ten neurogenic tumors, nine mixed tumors of the lacrymal gland and fourteen odd types.

Unilateral lesions confined to the anterior portion of the orbit are in the province of the ophthalmologic surgeon. The operative approach is by an anterior route. Reese describes a temporal transconjunc-

tival approach suitable for laterally placed anterior growths. For those placed mesially a similar incision on the nasal side is chosen. Depending upon the site of the growth, Davis has recommended exposure through the skin at the lower margin of the orbit and Benedict one for superior tumors through the brow.

The Krönlein operation common to surgeons of sixty years ago still has followers. Its awkwardness, the visible scar which is produced, often with deformity, its inadequate decompression and the inability to extend the exposure to intracranial structures if need be leave little to recommend it and it has rightly fallen into disuse. When there is evidence that the mass is posteriorly placed or with a possibility of intracranial extension, the transcranial approaches to be described are necessary.

In 1941, Dandy published a monograph on orbital tumors. Twenty-four operative cases were reported in which he practiced a transdural operation. Surgical exposure was obtained through a frontal bone flap. The dura was opened, the brain retracted and access to the orbit was gained through the dural covering at the base and the bone of the orbital plate. This approach was necessary for most of the growths in his series. The extensive intracranial involvement required opening of the dura for removal of the growth and the invaded bone and dura at the base and into the orbit. However, Dandy used the same method and route for certain tumors which were entirely confined to the orbit. For growths confined to the posterior portion of the orbit and for decompression in bilateral exophthalmos a transcranial but extradural operation has obvious advantages.

In 1915, the writer operated upon a patient with oxycephaly who was rapidly losing vision. By a transcranial but extradural route the orbit was opened and decompressed and the optic foramen unroofed with subsequent improvement. In 1931, a similarly performed orbital decompression for bilateral exophthalmos was carried out as an extradural bilateral procedure through a coronal incision with unroofing of the optic foramen and incision of the annulus of Zinn. Surgery for bilateral exophthalmos has been required in conditions due not to neoplasm but to general systemic disturbances associated with increases in the orbital content. Since then, modifications of this operation have been made, particularly by increasing the extent of the bone removal. The wide exposure of the orbital contents and the familiarity gained by these experiences have been of help when applied to patients with unilateral tumors, for the technical methods are the same.

FACTORS IN INTRA-ORBITAL TENSION

Decompression may be required because of dangerous progression in bilateral exophthalmos and because of our inability to remove the cause of pressure. Comparison of orbital decompression with decompression for intracranial tumor does not hold for one large group of conditions, viz., progressive exophthalmos associated with thyroid disease. In these, one is not dealing with neoplastic disease nor any single gross removable lesion. The increase in orbital content is dependent upon hormonal and constitutional disturbances. Various structures, viz., muscle, fat and fibrous tissue, are all affected. Much information has been gained in recent years regarding the alterations in the amount of fat and fluids present and the myopathy, muscle infiltration and change in muscle volume in the various stages of thyroid disease. In Basedow's description of exophthalmic goiter alteration in the orbital content, notably in the size of the extra-ocular muscles, was mentioned. That alteration in orbital structures was the important and perhaps the sole factor in the proptosis of thyrotoxicosis was lost sight of over the years by reason of misdirection of attention. Claude Bernard's discovery of the rôle of the sympathetics in animals in producing pupillary changes, alteration in the width of the palpebral aperture, protrusion of the globe and vasomotor manifestations was

associated with Müller's writings on the smooth muscles of the orbit. That Bernard's experimental findings in animals could correctly be applied to man was generally accepted. Not till long afterward was it appreciated that stimulation of the sympathetics in man, contrary to the happening in most animals, does not protrude the eye. The other responses produced in animals by Bernard occur in humans, but not the proptosis.

The erroneous assumption that the sympathetics caused proptosis in man was sufficiently satisfying that during the entire period of the development of our knowledge of the causes, physiology, pathology and treatment of thyroid disease, scant attention was given to the intraorbital changes and notes of pathologic studies at autopsy were almost nonexistent. In recent years, detailed observations have been made at the operating table by biopsies and also by postmortem examinations. Moreover, laboratory research has revealed the agents capable of producing tissue changes in the orbit. These changes produced experimentally are identical with those that occur in various stages of thyroid disorders.

By use of the exophthalmometer, which measures the position of the anterior surface of the cornea with reference to a fixed bony landmark, viz., the deepest concavity of the external orbital rim, a wide normal variation in measurement is demonstrated ranging from an infrequent reading as low as perhaps 11 mm. to 20 mm. or thereabouts. A fair normal average is about 16 mm. Measurements by expert examiners vary but usually not more than 1 mm. A large series of patients with thyrotoxicosis will average about 1.5 mm. greater than normals. Contrary to earlier impressions, it has been our experience that there is further protrusion of 1.5 mm. or more in over 40 per cent of thyrotoxic patients after thyroidectomy. Recession occurred postoperatively in about 5 per cent and the remainder showed no alteration. Contrary clinical impressions are due to postoperative lessening of the stare and decrease of lid retraction.

Our principal responsibility is in those instances when the exophthalmos becomes markedly progressive, particularly in the small number in which it attains a degree which endangers the patient's vision or life. The exophthalmometer readings in patients requiring operation have ranged from 22 to 35 mm. Such a measurement alone, without consideration of other factors, is no guide to the necessity for surgical treatment. In patients under observation, frequent measurements are essential to determine the progression. A full appreciation of the changes in orbital content due to alteration in solids or liquids is essential to an understanding of the surgical problem and the attendant difficulties in treatment.

From the pathologic and experimental contributions to our knowledge in recent years it is known that at different stages in the progress of proptosis there are altering proportions of orbital fat, fluids, muscle volume and lymphorrhages in the orbital tissues. Experimentally, these changes can be reproduced in animals and in fishes by injection of the thyrotropic hormone of the anterior pituitary body. All details of the complex inter-relationships of the glands of internal secretion which are involved are by no means settled.

In thyrotoxicosis, exophthalmos may be absent and other evidences of intoxication may be most severe. Again, the severity of the proptosis may be the principal concern and present the most urgent need for treatment while the nervous and circulatory disturbances may be insignificant. It is notable that severe and progressive exophthalmos may appear sometimes before other evidences of thyroid disease although progression most commonly is seen with normal or low basal metabolic rates and in postoperative patients. Progression of the protrusion by no means indicates overactivity of the gland but usually the reverse. Proptosis may appear before thyroid disease is apparent and proceed during

the stage of hyperactivity, continuing after thyroidectomy and after the establishment of a hypothyroid state. It occurs in extremes ranging from severe thyrotoxicosis to frank myxedema. It is prone to affect those in middle age and men more often than women. Most often it appears within some months after thyroidectomy.

The predominance of ocular manifestations in certain patients has been so striking and without altogether satisfactory explanation that it has afforded occasion for various recommendations in the classification of cases and for more or less complex theorizing on causation. The literature has grown rapidly under such headings as progressive exophthalmos after thyroidectomy, malignant exophthalmos, exophthalmic ophthalmoplegia (Brain), hyperophthalmopathic thyrotoxicosis (Means) and thyrotropic exophthalmos (Mulvany).

To the surgeon responsible for orbital decompression in the case where proptosis has not halted either spontaneously or with medical treatment the problem would be simple if the orbital tension were due to a solid mass. Allowing additional space would permit recession of the dangerous protrusion. Unfortunately, we are dealing with more than an increase of solids. There is a variable and fluctuating amount of fluid in the tissues dependent upon and governed by biochemical factors. The degree of retrobulbar tension and protrusion is determined by this fluid pressure and resistance of the globe to the protrusion. In the most advanced stages of malignant exophthalmos, there is an enormous increase in fluid content with tremendous extrusion of the edematous mucosa from the sclera and lids. When the tendency for fluid accumulation is so pronounced, orbital decompression allows some space for expansion and lessens retrobulbar tension but does nothing to alter the underlying biochemical factors. These persist and when allowed more space by decompression the tissues take up still more fluid.

It is for this reason that treatment by orbital decompression should not be delayed until the final stage of malignant exophthalmos is reached. On the other hand, decompression is not to be regarded as a cosmetic operation. In a few instances, bilateral progressive exophthalmos has appeared with no discoverable thyroid relationship nor other systemic disorders such as Schuller-Christian disease or hypertension. In the vast majority some connection with thyroid disturbance is clear. Usually some months after thyroidectomy these patients seek treatment although it may be many years later. As protrusion increases swelling of the lids appears. This is earlier and more marked in the upper than the lower lids. From the work of Pochin it appears that this is due to fat accumulation. Impaired motility and diplopia appear at variable stages. Photophobia, lacrimation, a watery-appearing scleral conjunctiva, scleral injection and then protrusion of the mucosa from between the lids follow. Closure of the lids is incomplete. Desiccation of the cornea, ulceration, opacities and panophthalmitis result. Before adequate treatment was available the patients in this severe state succumbed to meningitis or brain abscess.

Those patients in whom vision is threatened by corneal exposure and those in whom vision is diminishing for other reasons are suitable candidates for decompression. Vision may be threatened also by choking of the optic nerve or by pressure on the optic nerve at the optic foramen. In a few instances altitudinal hemianopsia has been attributed to this latter cause. It has been relieved by orbital decompression without unroofing of the optic foramen. Frequently, there is complaint of diminishing vision without visible alteration in the cornea or media, without choking or perimetric field changes and without a satisfactory explanation to be found in the appearance of disc or retina. Restoration of visual acuity follows decompression. Such manifestations appearing with an increasing degree of exophthalmos constitute the operative indications. Impairment of extra-

ocular movements is likely to be noted first in the upward gaze or laterally with no impairment of downward motion. In the absence of other indications, we have not considered impaired movement of the globes a sufficient reason for decompression. Often, however, it is associated with other urgent manifestations.

During the period of progression the use of thyroid substance and iodine is generally believed to be of advantage and is worthy of trial. Water excretion and lessening of water storage are promoted. X-ray therapy of the pituitary gland or of the eye muscles themselves is of unproved value although it has some advocates. Continued progression with evidence of beginning scleral edema is a danger sign and warrants immediate consideration of operative intervention.

When decompression is required, the widest possible bone removal is none too much for the desired result. Restricted procedures by the intracranial route, the Krönlein method or operation externally through brow or lids have been unsatisfactory, or if helpful have been performed in the less dangerous cases or for cosmetic indications.

TECHNICAL CONSIDERATIONS

The technical points involved in orbital decompression have become familiar to neurologic surgeons but emphasis on some features may not be amiss. These are applicable especially to those cases of endocrine origin but to a considerable extent are applicable to all extensive orbital exposures.

The preoperative and operative management for unilateral and bilateral cases are similar. Preoperative x-ray studies showing the extent of the frontal and ethmoid sinuses into the orbital plate aid one in avoiding any inadvertent opening into them.

As a first step three fine mattress sutures are used to barely approximate the lids and are lightly tied after generous lubrication of the lids with boric ophthalmic ointment. With care, no lashes are turned in. Such closure prevents subsequent protrusion of the mucosa if it becomes edematous or if edema already present increases. If corneal damage is already present, a tarsorrhaphy is in order.

When dealing with posteriorly placed tumors, with those that are both intracranial and intraorbital and for orbital decompressions, several procedures are used. For unilateral exposures we have used a scalp incision as illustrated in Fig. 13. The resulting short, midline scar is scarcely visible. A small quadrilateral bone flap is turned with the hinge in the temporal fossa. For bilateral decompression a coronal or Souttar scalp incision is made and the scalp dissected forward to the brow. The larger frontal bone flaps as used originally (Fig. 2) have been replaced by smaller ones which are ample or by triangular bone flaps having a trephine opening which is common to both in the midline as used by Poppen. (Fig. 3.) After reflection of the bone flaps the dura is peeled back from the orbital plate. For decompression the extent of the bone removal from the orbit is greater than may be required for an intra-orbital tumor. It should be wide and limited only by the extent of the frontal sinuses anteriorly and the ethmoids medially. (Fig. 4.) The posterolateral wall of the orbit is removed close up to the orbital rim after retraction of the temporal muscle. This excision is carried downward close to the antrum. Behind this the pterion and sphenoidal ridge are rongeured away. During the bone removal care should be taken to avoid opening the thin periorbita. If this is ruptured, the persistent herniation of the tense orbital content hampers the operator. It has been found unnecessary to unroof the optic foramen and incise the annulus of Zinn as was reported in our earlier patients.

Upon completion of the bone opening, hemostasis is obtained and the periorbita widely opened to the limits of the exposure. (Fig. 5.) The tumor removal or the decom-

pression with a small muscle biopsy is concluded. Upon closure the orbital content will be in contact with the frontal dura above, the dura of the temporal lobe behind and the temporal muscle laterally.

a small stab incision posterior to the operative incision and above the ear. This is removed in twenty-four or thirty-six hours.

The bone flaps are replaced and fastened in position and customary closure in layers

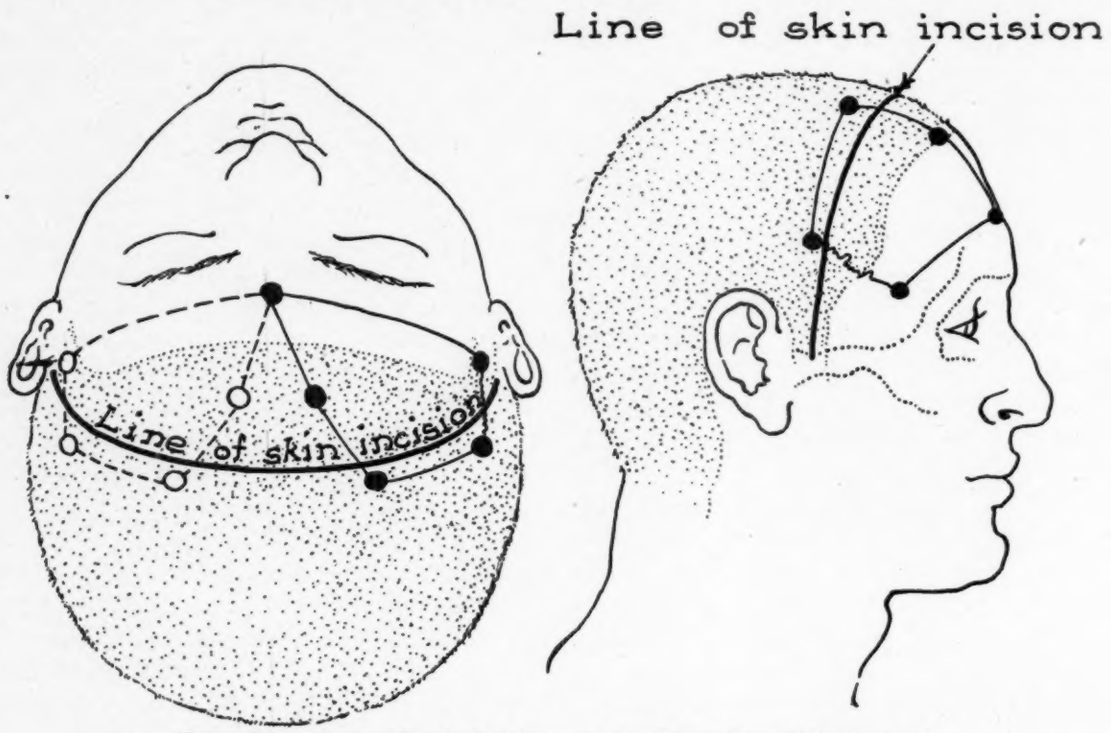


FIG. 2. Line of coronal scalp incision; outline of bone flaps originally used.

Hemostasis should be detailed. There is, moreover, less edema if a small, sewn, rubber drain is led from the pterion back beneath the temporal muscle and through of the temporal muscle, fascia, galea and skin is made. Postoperatively, additional lubrication is applied, a cover of gutta percha tissue and a pad of suitably shaped

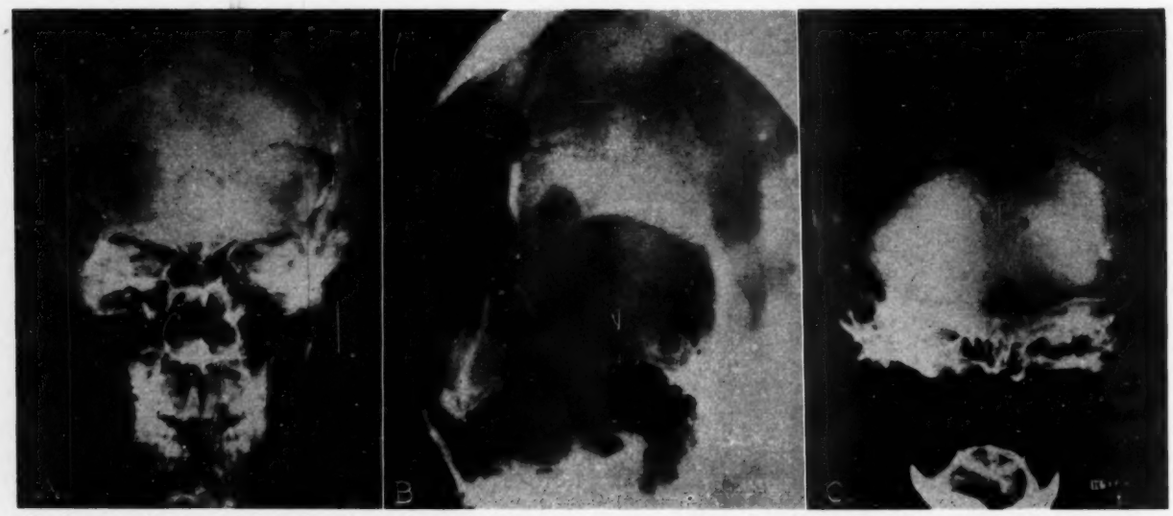


FIG. 3. A, triangular bone flaps introduced by Poppen for use in bilateral decompressions; B, showing unroofed optic foramen; C, large bone flaps as originally devised.

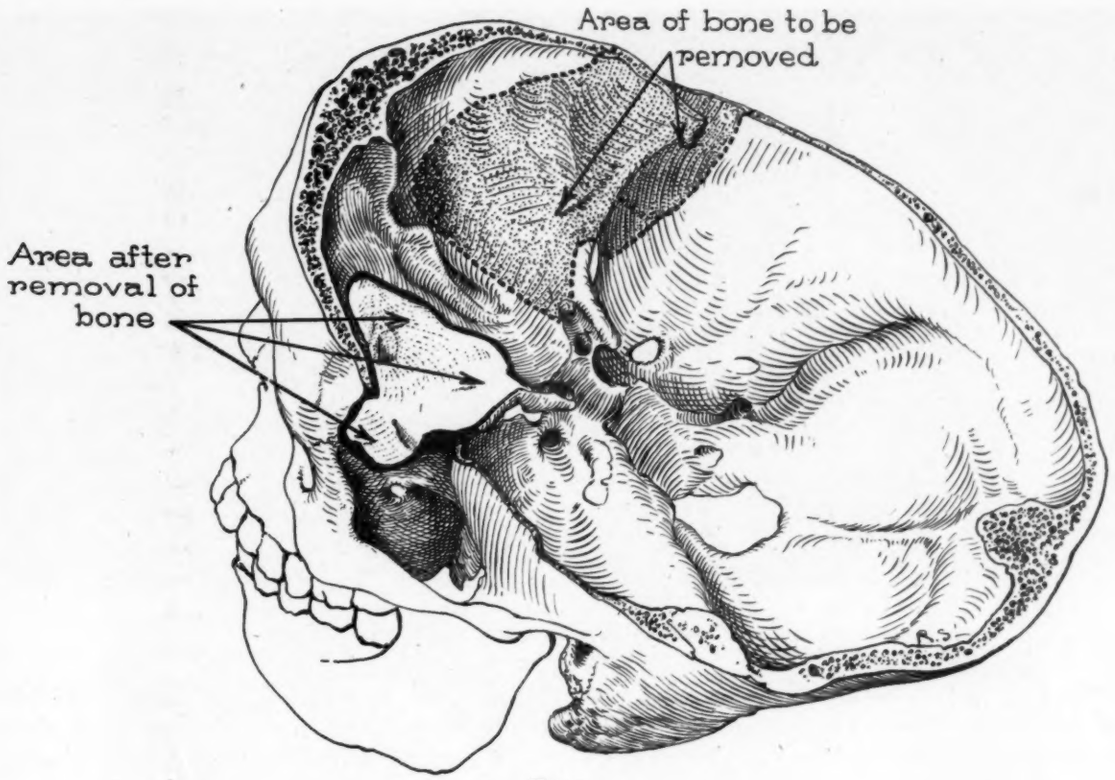


Fig. 4.

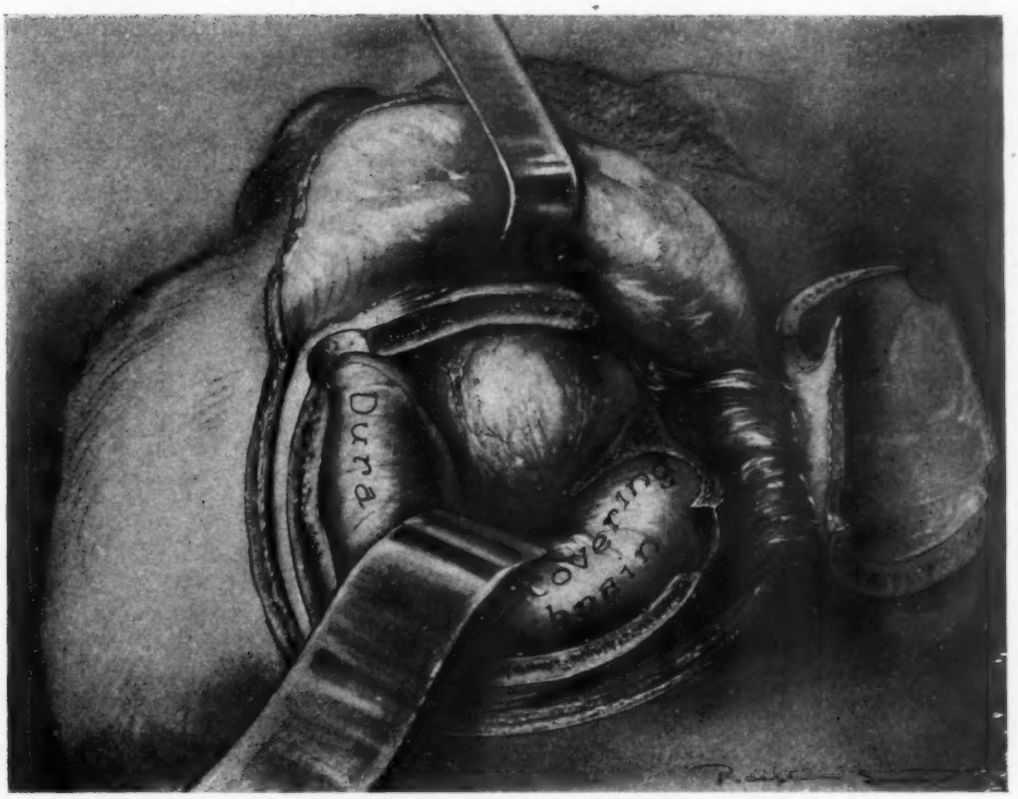


Fig. 5. Bone removal completed; periorbita not yet opened.

A B C

FIG. 6. A, B and C, advanced exophthalmos; preoperative state.

sponge rubber are placed over the lids and light compression is maintained with an Ace bandage over the head dressing. This compression is continued and the sutures are usually left in place for about a week. Before that time temporary removal of the dressing and cleansing with boric acid solution are done to remove mucoid secretion. If the mattress sutures have not been pulled and tied tightly, there should be no infection about them.

The postoperative treatment, barring such complications as may be common to all frontal operations, is simple or prolonged depending upon the protrusion and edema of the scleral and palpebral mucosa. Ordinarily, and with the precautions mentioned, there will be little trouble if there has not been some preoperative extrusion. If edema is present and pronounced, it is apt to be continued for some time and require a prolonged hospital stay. Care of edema must be closely supervised by medical personnel. Removal of secretion with 2 per cent boric acid solution and lubrication with boric ophthalmic oint-

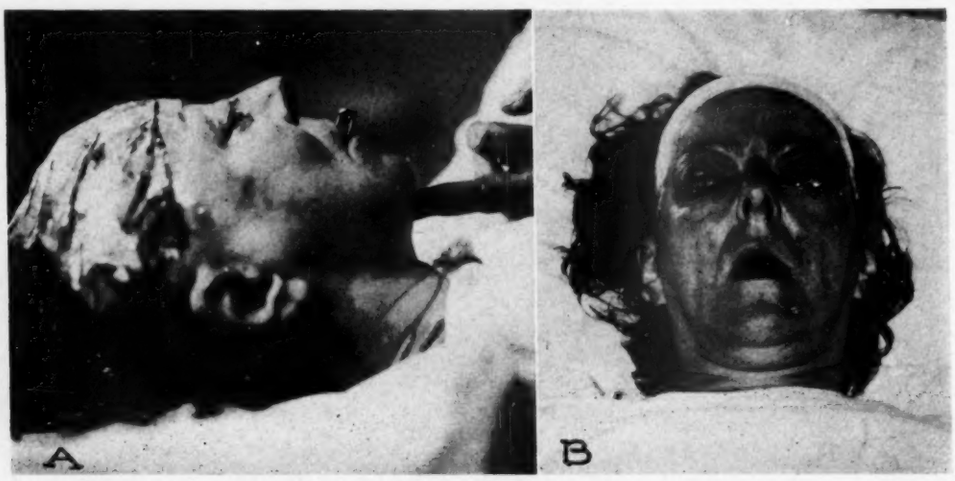


FIG. 7. Postoperative stages of patient shown in Figure 6. A, recession of globes at conclusion of operation; B, protrusion of edematous mucosa one week later.

Fig. 7. C and D, condition at end of four weeks; E and F, one year after operation.

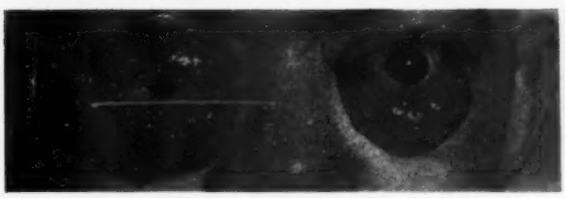


Fig. 8. Edema in unoperated patient.

FIG. 9. A and B, preoperative state; protrusion and scleral irritation and beginning edema; C and D, postoperative result.

ment are carried out with avoidance of all irritating washes or drops. A goggle or perforated shield prevents drying. Resuturing of lids after replacement of the mucosa may be needed. Limitation of fluids and salt, use of thyroid substance and spinal puncture are tried but the results of these measures are not impressive.

Immediately following operative decompression, the ocular recession is marked. Pulsation of the globe transmitted from the dura is readily evident. Shortly, due to edema, there will be return of some protrusion. The pulsation will diminish but will persist in some measure over a period of months. The patients are rarely aware of it and have no visual disturbance as a result.

Swollen optic discs and retinal hemorrhages are present in the minority of patients presenting themselves for operation. In our first experiences, it was thought advisable to unroof the optic foramen for relief of the choking. Later experiences show this is unnecessary. The swelling disappears rapidly after decompression.

Permanent recession of proptosis of from one or two to several millimeters follows decompression. Progression has not occurred postoperatively in our patients. Impaired ocular movements improve markedly but some fault in movement and diplopia may persist.

Bilateral orbital decompression may be performed at one operation and this has been done in most of our subjects. It involves a longer operative procedure and might be criticized on the greater risks of a complicating infection. When great edema

A B

FIG. 10. A, preoperative condition; B, postoperative result.

Fig. 11. Preoperative appearance and postoperative result three weeks later.

of the mucous membrane is present with or without corneal ulceration and considering the period with eyes bandaged, a unilateral operation is sometimes decided upon.

In the postoperative period, edema of the scleral and palpebral mucosa with protrusion from between the lids has been the most frequent concern. When present before operation, edema is apt to be exaggerated for a postoperative period. This applies to patients with progressive bilateral exophthalmos. (Figs. 6 and 7.)

Few other complications have been met with in thirty-nine cases of this type. Rhinorrhea occurred in three patients due to opening of the ethmoids. In one instance, a pneumococcic meningitis followed but was controlled promptly by chemotherapy. In all patients the rhinorrhea disappeared in a few days. Marked retraction or elevation of the dura may be followed by frontal lobe signs such as disorientation and confusion over a short time. In this series there have been no deaths. In a few patients operation on one side only was required.

Operation for unilateral intra-orbital tumor differs in no essential way from that described above for bilateral decompression. A small quadrilateral frontal bone flap is preferable to a triangular one and orbital bone removal need not be as extensive. Suturing of the lids prior to operation and a light compression bandage following it are helpful.

Since the transcranial extradural operation was devised, other methods of decompression have been suggested and reported in small series of cases. Kistner has decom-

A B

Fig. 12. A and B, examples of so-called "exophthalmic ophthalmoplegia"; impaired motility, moderate protrusion; no evidence of thyroid disturbance.

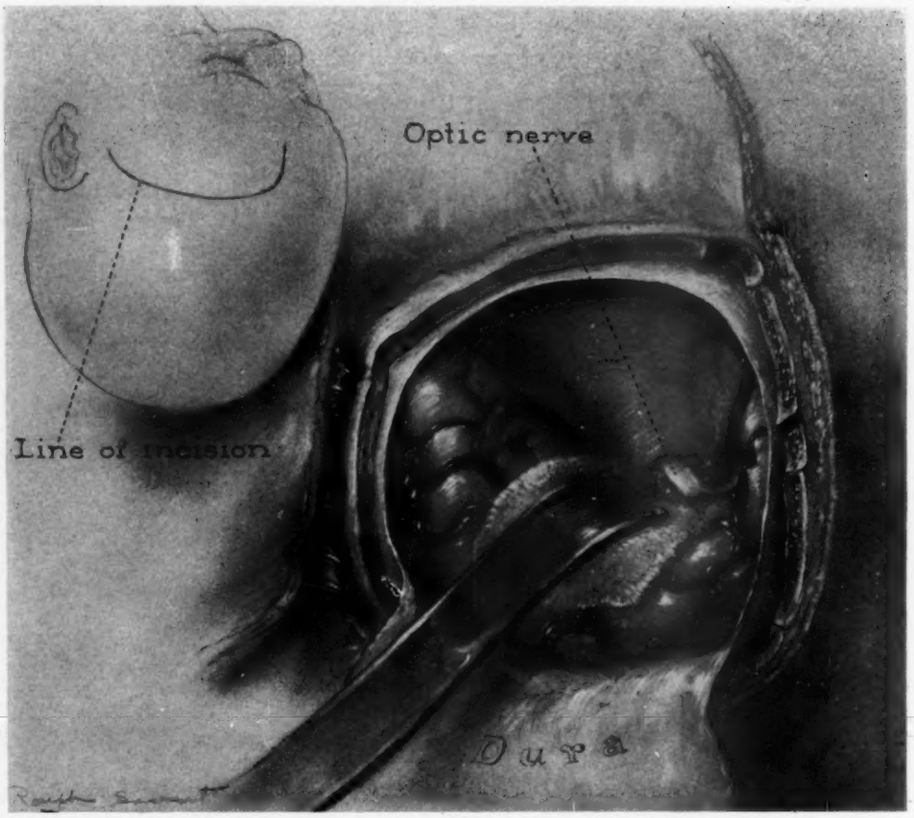


FIG. 13. Frontal pole retracted exposing orbital plate, sphenoidal ridge, optic nerve and chiasm.

pressed the orbit into the frontal sinuses with good results. Bothman, Sewall, Swift and others have practiced or advocated removing the bony wall of ethmoids or antrum for the same purpose. The writer has had no experience with these. In our patients the largest decompression possible has been sought through a clean field. The risk has been minimal and the procedure should leave no visible scar or deformity.

Guyton, in a discussion of decompression of the orbit, advocates a skin incision through the hair line and a comparatively small lateral or posterolateral decompression of the orbit. Such an opening in our experience, although restricted as a decompressive operation, serves a valuable purpose. He recommends it as suitable for cosmetic purposes as well as for the more dangerous proptosis with which we have dealt.

For combined intra-orbital and intracranial lesions one of two courses may be followed. A larger bone flap than the small triangular one is required. If the orbit is explored first, the intracranial lesion may be pursued by opening the dura along the sphenoidal ridge. If an intradural growth is known preoperatively to be present, the dura is opened over the frontal pole which is then retracted (Fig. 13) for a radical procedure and the concern is not primarily with the exophthalmic manifestation.

For exposure of the posterior and lateral portion of the orbit and visualization of the optic nerve and its neighborhood intracranially a temporal approach has a useful place. A scalp incision as shown in Figure 14 is made as well as a similar one in the temporal fascia and muscle. A small fringe remains attached to the upper margin of the temporal fossa for later resuturing. With a periosteal separator, the muscle is cleaved off from the temporal fossa and the bone removed. (Fig. 14 and 15.) By opening the dura and sucking out fluid

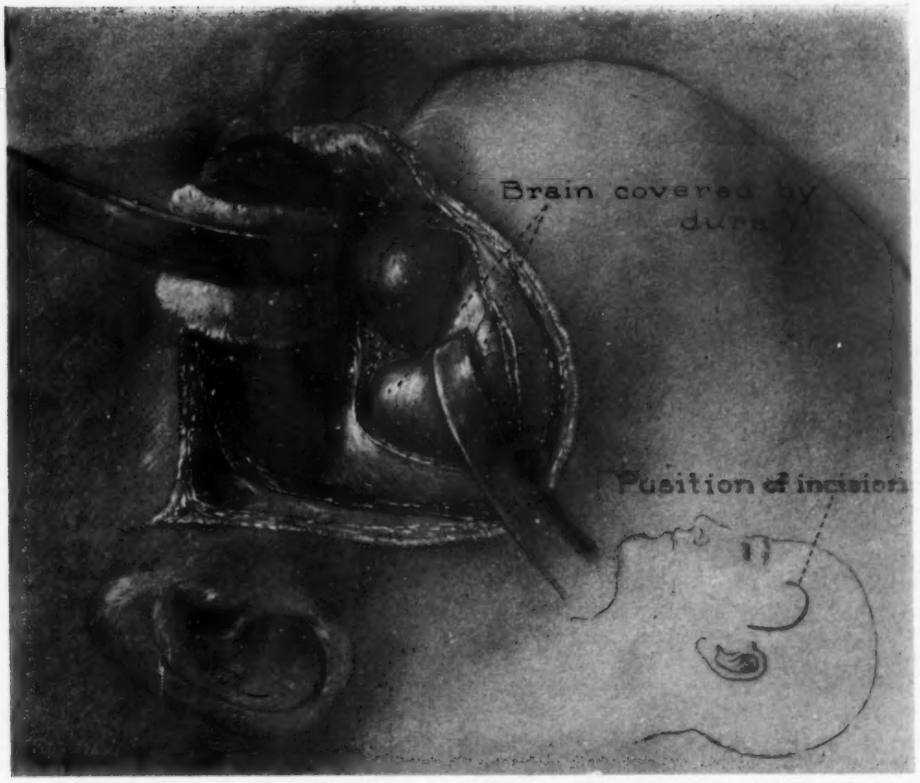


Fig. 14. Line of incision (insert); temporal muscle retracted; bone removal with periorbita and dura of the frontal and temporal lobes exposed.

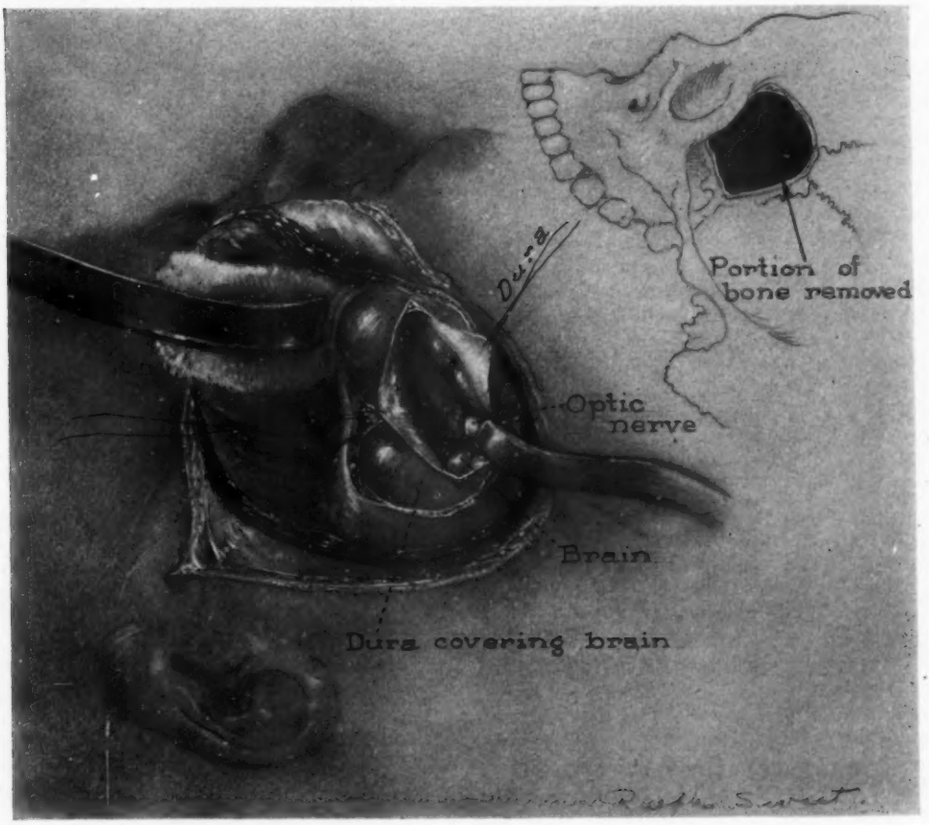


Fig. 15. Intracranial exposure through temporal approach.

intracranial exploration is readily made. This has been found useful in determining involvement of the optic nerve prior to intra-orbital enucleation. It has been highly satisfactory for the laterally placed tumors such as cholesteatomas.

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COMPLICATIONS ACCOMPANYING SURGICAL RELIEF OF PAIN IN TRIGEMINAL NEURALGIA

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THE diagnosis of major trigeminal neuralgia in the typical patient is easy. An elderly patient presents himself with sudden, intense, lancinating pain in one or the other side of the face. This pain usually affects the upper or lower lip and extends up to, but never across, the midline. The pain is brought on by any local irritation such as talking, eating or washing the face. The pain is definitely paroxysmal in character, coming on in intense, stabbing waves with little or no abnormal sensation between attacks in the area involved.

As a rule, the patient has had a short, initial spasm months or years before the surgeon sees him and, between subsequent attacks, has noted periods of spontaneous remission during which there has been no abnormal sensation of any kind in the face. The presence of a trigger zone is characteristic. For example, if the upper and lower lips are involved in the pain, it is frequently noticed that touching either area will cause a spasm of pain to pass from the lower half of the face into the upper half and the patient soon learns to avoid touching this trigger area. Not infrequently, the experienced observer makes the diagnosis of major trigeminal neuralgia as the patient steps into the office. He will notice that part of the face about the upper lip and the ala of the nose is unwashed, dirty and greasy. This is the trigger zone. Any attempt on the part of the patient to clean up this area results, at once, in the lancinating, stabbing, paroxysmal pain so characteristic of this disease.

Fortunately, both sides of the face are but rarely involved. In a series of, roughly, sixteen hundred cases of verified major trigeminal neuralgia, there have been but two in which the pain was noted to be bilateral at the same time. There have been forty-three cases in which, after the pain had occurred on one side, this attack was followed a number of years later by similar pain on the opposite side.

The treatment of major trigeminal neuralgia has become completely standardized. Section of the sensory root of the trigeminal nerve is the accepted method for producing permanent relief. Unquestionably the relief from the intolerable, stabbing, lancinating, paroxysmal pain characteristic of this condition is complete. Nevertheless, the patient is penalized for relief from this pain. Any wide experience with the results of the treatment of major trigeminal neuralgia by root section will soon convince the honest and competent observer that while, for the most part, section of the sensory root gives the patient the freedom from pain that he expects, nevertheless, he pays a certain price for complete relief of pain which, in some instances, is rather higher than had been anticipated.

The price that the patient pays for the complete relief of pain is total anesthesia in the areas to which the pain was originally referred. This anesthesia is produced by section of the sensory root, whether the root be cut at the pons by the occipital approach or just behind the ganglion by the temporal route. Dandy[1] claimed as one of the advantages of the occipital approach to the sensory root that, by this technic, he was able to cut just the lateral fibers of the root and thereby bring about a selective relief of pain alone, without loss of touch sensation. Our experience has been entirely at variance with Dandy's. No matter where the sensory root is cut, com-

plete loss of all modalities of sensation was produced if pain sensation was abolished. Occasionally, when a subtotal section of the root was performed, total anesthesia in the third division for all modalities of sensation and a partial loss of sensation in the second division resulted. However, in the second division, where sensation was partially preserved, it was always impossible to determine that pain sensation was lost to a greater degree than touch. It is quite true that the operation devised by Sjöqvist,[2] the section of the descending root of the trigeminal nerve in the medulla, has produced selective loss of pain sensation without an equivalent loss in touch sensation. We were enthusiastic about the possibilities of the Sjöqvist technic until experience showed that, unfortunately, the fibers of the third division, which is very commonly involved in most severe cases of major trigeminal neuralgia, lay most centrally in the medulla. A deep, bold section in the medullary area was necessary to assure section of these centrally situated third division fibers. The consequences of a deep section in this area was sometimes quite devastating for the patient. A very marked ipsolateral ataxia and dyssynergia of a permanent nature can be produced by too deep a section of the medulla in attempting to carry out the Sjöqvist technic. Medullary tractotomy was carried out in six patients with typical major trigeminal neuralgia. In five of these six patients, the pain recurred and root section was necessary to relieve the pain permanently. The sixth patient has been entirely comfortable for the past seven years. Very little, if any, loss of touch sensation is demonstrable in this patient although pain has been entirely lost and his major trigeminal neuralgia completely relieved.

One important consideration exists when the surgical treatment of major trigeminal neuralgia is suggested. It must always be remembered that facial neuralgia is not a lethal disease. It may make the patient extraordinarily uncomfortable and unhappy but as far as our records show, there have been only two cases of suicide because of this dreadful facial pain in our series of some sixteen hundred patients. Consequently, when surgery is considered the procedure used for relief of pain must be that which in the surgeon's hands carries the lowest mortality. As has been stated, there are two routes of approach to the trigeminal afferent fibers; one, through the temporal fossa, either intradural or extradural, with section of the root immediately posterior to the gasserian ganglion; and secondly, through the posterior fossa, either cutting the sensory root where it leaves the pons or severing the descending fibers of the trigeminal nucleus in the medulla. Either of these surgical approaches can be carried out with the patient in the sitting position. It is certain that the transtemporal approach lends itself a little more readily to the use of a local anesthesia than does the occipital approach. Dandy,[1] who originated the occipital approach, carried his procedure through with the patient in the prone position. When it is remembered that 85 per cent of the patients who are operated on for major trigeminal neuralgia are over sixty years of age, with all the possible complications accompanying advanced years as hypertension, arteriosclerosis, myocardial or renal disease, the disadvantages of the prone position, with the need for endotracheal or rectal anesthesia, at once become obvious. The exposure of the sensory root by the occipital approach is rather more difficult than by the temporal approach. The root lies more deeply. If bleeding occurs, consequent upon rupture of the petrosal vein which runs from the cerebellum to the lateral sinus directly in the line of this approach or if, as is not uncommonly the case, an artery accompanies the sensory root and is injured at the time the root is sectioned, the control of hemorrhage in this deep field is a matter of considerable difficulty. Electrosurgery and the use of fibrin foam and other coagulants have rendered the hazards accompanying hemorrhage much less great. The close proximity of the brain stem and

the facial nerve makes the careful and conscientious operator very nervous if he finds himself involved in bleeding in this dangerous area. A medullary tractotomy certainly cannot be carried out under local anesthesia. When the incision is made into the medulla to section the descending root, the patient suffers intense, lancinating pain and is quite likely to jump and twist. The depth to which the incision into the brain stem should penetrate is a matter of millimeters. Any sudden movement on the part of the patient can very well involve him in serious consequences in spite of the best efforts of the operator. It is our very distinct impression that the erect position is the safest for section of the sensory root, whether this be carried out through the temporal or through the occipital approach. Certainly the consecutive series of cases operated on by Frazier[3] and Cushing[4] without a mortality has never been surpassed. However, their success was not due necessarily to position because Cushing operated on his patients in the lateral, horizontal position whereas Frazier always employed the sitting position. Both of these operators used very light, open drop ether, given by nurse anesthetists who were skilled and experienced in carrying these patients through this operative procedure with a minimum of anesthesia. The modern, young, medical anesthetist knows how to give a dozen different types of anesthesia but, in our experience at least, he is deficient in his knowledge of the value of small amounts of open drop ether. In sensory root section no relaxation is necessary. All the operator asks of the anesthetist is to keep the patient sufficiently under the anesthetic so that he does not move. As soon as the root is cut the anesthesia can, of course, be stopped. We have seen skilled nurse anesthetists carry elderly patients along for two hours on less than 3 ounces of ether and have them so lightly anesthetized throughout the operative procedure that they would respond during the closure of the wound. It is difficult to persuade the modern, medical anesthetist to give open drop ether in this fashion.

Recent experience has shown that elderly patients who have been given morphine preoperatively are likely to suffer a serious fall in blood pressure when placed in the erect position. There has been at least one recent fatality in our clinic which we attributed to the preoperative administration of ¼ gr. of morphine. Consequently, we have recently eliminated morphine as a preoperative medication, substituting a small dose of phenobarbital plus atropine. Our overall mortality in major trigeminal neuralgia, using the erect position and the transtemporal extradural approach, is 1.85 per cent. We would repeat that since major trigeminal neuralgia is not a lethal disease, if an operative death occurs in an attempt to relieve it, the surgeon has unquestionably predeceased the patient unnecessarily. It is for this reason that we insist that every precaution should be taken to protect these elderly patients who have major trigeminal neuralgia from any operative accident, whether that accident be due to the surgical technic used or to the anesthesia employed.

A second complication, which can mar the results of section of the posterior root for relief of the stabbing, lancinating pain produced by major trigeminal neuralgia, is a paresthesia of the face in the anesthetic area. In our series, 62 per cent of the patients were completely and entirely satisfied with the operative results. Thirty per cent were very glad that they had had the operation and had been relieved of their pain but stated that they were conscious of the numbness of the face. They accepted this numbness quite philosophically as the price they had to pay for relief of pain. The remaining 8 per cent complained of the numbness in their face; about one-half of these, or 5 per cent of the total, complained with very considerable bitterness. The face burns, it has a creepy, crawling sensation in the numb area; the tongue and lip feel as though they had been dusted with pepper and are hot and raw.

These patients insist that the operation, while not a complete failure, has not by any means lived up to their expectations as far as relief from pain is concerned. An occasional patient will state that he is more uncomfortable after operation than he was before because this burning sensation in the face is present for twenty-four hours a day and keeps him awake at night, whereas the lancinating pain was only intermittent, rarely occurred at night, and between attacks, which he could avoid by precautions to which he had become accustomed, there was no change in facial sensation. The possibility of paresthesia in the anesthetic area is the principal objection to sensory root section. It was for this reason that we hailed with so much hopefulness the reports of medullary tractotomy described by Sjöqvist, with its production of selective analgesia without complete anesthesia. However, we have become completely dissatisfied with the Sjöqvist procedure because, in our experience with six patients with major trigeminal neuralgia, two complained of paresthesia and five had eventual recurrence of the trigeminal pain.

It is our firm opinion that the best method for preventing paresthesia in the face following root section is by preliminary relief of the neuralgic pain by alcohol injection of the appropriate branch, or branches, of the nerve involved. We appreciate fully that preoperative block of the second or third division with alcohol is a painful ordeal for the patient. The nerve cannot in every instance be accurately injected and the relief produced is not permanent. However, following a successful block, the patient can be relieved of pain for a year to eighteen months and a second block will keep him pain-free for another year. More important, the anesthesia resulting from the alcohol block is precisely similar to that which will be produced by section of the sensory root. If, therefore, a patient has had his pain relieved by alcohol block, he knows how his face will feel and appreciates very fully the price that he must pay for permanent relief of pain. After the anesthesia, consequent upon alcohol block, wears away, the pain recurs. When the patient returns asking for complete relief, he may then be told that he can be permanently relieved by root section but that he will always have the anesthesia. If, during temporary relief by block, the anesthesia has not been an annoyance to him, he is quite willing to have the sensory root cut and the anesthesia made permanent. If, after an alcohol injection, he objects to the numbness of his face, he can be told that if he will be patient for a year that numbness will disappear, sensation will return to normal and his pain will recur. However, once the sensory root has been cut, the anesthesia produced is permanent and if the patient objects to it nothing can be done to improve the situation. It is for this reason that we believe that preparatory alcohol injection, in spite of its disadvantages, has a very definite place in the treatment of major trigeminal neuralgia. Our experience has been that those patients who have had preliminary block of one or the other branches of the nerve are much less likely to complain of the permanent, total anesthesia which follows section of the sensory root of the nerve and consequently are less likely to develop burning paresthesia and other disagreeable sensations in the anesthetic area.

As far as the first and second divisions are concerned, other methods for temporary relief of pain are available than simple alcohol injection. Avulsion of the supraorbital nerve through an incision in the eyebrow is a simple, safe procedure and will relieve the supraorbital neuralgia for four or five years. An incision through the mucous membrane under the upper lip will expose the second division very readily. Once the infraorbital foramen with the emerging nerve is visible, it is a simple matter to introduce a wire for 2 or 3 cm. into the infraorbital canal and coagulate the nerve with the electrosurgical unit. In a nervous patient who insists upon an anesthetic for relief of second division neuralgia, this procedure has some ad-

vantage over direct alcohol injection. As far as the third division is concerned, if the lower lip and teeth alone are involved, the nerve can be blocked with a drop or two of alcohol where it descends around the ramus of the mandible. If the tongue is involved, however, the lingual branch of the third division usually leaves the main trunk above this point. Consequently, alcohol injection through the cheek, picking up the whole nerve where it leaves the skull is necessary when the paroxysms of pain involve the tongue.

The third complication of section of the sensory root, particularly when it is completely sectioned, is keratitis and ulceration of the cornea in the ipsolateral eye. Subtotal avulsion of the sensory root, as suggested by Frazier[5] in 1925, sparing that part of the root that runs to the first division and thus not completely desensitizing the cornea, has well nigh eliminated this particular complication. The problem always arises, of course, in those patients who have major trigeminal neuralgia in all three divisions, as to whether a subtotal avulsion is appropriate. It is our definite experience that it should be done unless the supraorbital area is the trigger zone. Fortunately, this is the exception rather than the rule. In those instances when the trigger zone lies in the second and third division and when irritation of this trigger zone results in pain in the first division, it is entirely safe to carry through a subtotal avulsion. Desensitization of the trigger zone will eliminate the spread of pain into the first division. However, in certain cases when a subtotal avulsion has been done, there is, later on, a spread into the supraorbital area. If this occurs, it is a simple matter to avulse the supraorbital nerve at its foramen. It is, in our opinion, always much better to preserve the fibers in the root running to the first division than to do a total section. As we have said, if pain spreads later on into the first division, it can be relieved for four or five years by avulsion of the supraorbital nerve. Many of these patients are elderly and much can happen in five years. In our series there have been between 5 and 10 per cent of recurrences in the first division, usually after six or seven years have passed. It is much better, in our opinion, to accept the risk of recurrence in the first division consequent upon sparing the inner fibers of the sensory root than it is to avulse completely the sensory root and risk the possibility of complications in the cornea.

Prior to 1925, when total avulsion of the sensory root was the routine procedure, the incidence of some degree of keratitis was 15 per cent. In about one-half of these patients, closure of the lid was necessary to control the keratitis and in between 4 and 5 per cent of the total patients the eye on that side was lost. This calamity usually followed neglect of the eye on the part of the patient and attending physician. The keratitis spread, burrowed in to involve the anterior chamber, produced an anterior hypopyon and enucleation of the eye was necessary. At the present time most competent ophthalmologists recognize this situation promptly and close the eyelid. Closure of the eyelid will result in healing of the keratitis within ninety-six hours. Tarsorrhaphy is the only proper treatment for this complication.

The final complication which may accompany operation on the sensory root by either the temporal or occipital approach is facial paralysis. In the last five hundred patients operated on through the temporal approach, this complication has appeared in, roughly, 6 per cent of patients. Fortunately, in none of these patients has the paralysis been of a permanent nature. In every instance it has disappeared within six months. When the sensory root has been completely avulsed, the appearance of a facial weakness creates a very awkward situation. Inability to close the ipsolateral eye following complete section of the root, with total corneal anesthesia, will certainly result in drying of the cornea and consequent ulceration. Closure of the lid is therefore necessary in all these patients. The lid should be kept closed until

there has been sufficient return of power in the facial nerve to assure closure of the eyelid. If subtotal avulsion has been done and if corneal sensitivity is retained, tarsorrhaphy is not necessary. However, very close watch should be kept on the eye and if there is any slightest evidence of keratitis, lid closure is imperative.

The cause for facial paralysis or weakness following approach to the sensory root by the temporal extradural route is injury to the superficial petrosal vein or the vidian nerve. This nerve and vein run from the facial canal along the anterior face of the petrous ridge and under the ganglion. For this reason during the separation of the dura from the ganglion sheath, it is necessary to take care that the dura is not stripped from the anterior surface of the petrous ridge. The dissection should be made in such a fashion that the root can be exposed without elevating the dura in this area. If the dura covering this nerve and vein is not disturbed, facial paralysis is much less likely to result. The avoidance of facial paralysis during an approach to the sensory root through the temporal fossa is one of the claims made for the intradural approach. Facial paralysis or weakness can also follow the occipital approach to the sensory root. However, during this approach it is rather more likely that the facial nerve is seriously injured with a greater probability of permanent and lasting facial weakness.

In summary, it can be said that while section of the sensory root will relieve permanently and completely the savage, paroxysmal pain of major trigeminal neuralgia, nevertheless, both surgeon and patient must realize that for relief of pain the patient pays a definite price. The basic, inescapable price that he pays is complete anesthesia of the face. The surgeon's skill in selecting patients for operative procedure and his deftness in carrying out the surgical technic can eliminate all of the other complications herein enumerated. It is important, however, for both surgeon and patient to realize that, while the operative procedure for the relief of major trigeminal neuralgia can and will relieve the paroxysmal pain, nevertheless, the pathway to complete satisfaction with the operative procedure on the part of the patient is strewn with certain definite hazards.

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HYPERTENSION AND ITS SURGICAL TREATMENT BY BILATERAL SUPRADIAPHRAGMATIC SPLANCHNICECTOMY*

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HYPERTENSION presents the most serious medical problem before us today. No other disease exacts such an appalling annual toll. Its final manifestations of either cerebral, cardiac or renal damage account for more than 200,000 deaths each year in our country alone. Few realize that nearly all cases of so-called cerebral accidents, which include both intracranial hemorrhages and cerebrovascular thromboses, most cases of heart disease, including those with coronary thrombosis, and the great majority of cases of so-called Bright's disease have as their primary factor, hypertension.

The financial loss due to necessarily curtailed physical and mental activities resulting simply from the distressing symptoms of hypertension, and the actual loss of productive time due to the semi-invalidism of many victims of hypertension reach staggering figures. When evaluating the enormity of this disease, we must not forget the amount of suffering it causes, headaches, nervousness, irritability, insomnia, nocturia, shortness of breath and the all too frequent clouding of mentality. Any treatment which, with reasonable safety, can reduce this enormous death toll or can alleviate in a worth while degree the distressing symptoms and prolong the life of these patients is certainly worth while. Medical treatment has signally failed in many cases and we believe that surgery today offers far more than any other type of therapy a real hope of attaining these ends.

Considerable confusion has arisen because of the various designations of hypertension. We prefer the term arterial hypertension but commonly the terms essential or benign hypertension are used. Both are misnomers because it is not essential, as was at one time believed, to have an increased blood pressure in order to force blood through a possibly inadequate arterial system; and the disease is never benign although serious manifestations may not appear for many years.

The blood pressure may be temporarily or continuously elevated in a wide variety of conditions, for example, in cerebral trauma the blood pressure may rise very rapidly with the increase in intracranial pressure either from hemorrhage or edema. Certain tumors as basophilic adenomas of the pituitary gland and some adrenal tumors can produce either a continuous or recurrent elevation of blood pressure and rather late manifestations of chronic nephritis may also produce hypertension. Coarctation of the aorta, while uncommon, should always be considered a possibility in any patient, especially in a young individual who is found to have hypertension. This is a very brief list of the possible causes of elevated blood pressure. Such a list comprising forty-seven pathologic conditions associated with an elevated blood pressure was compiled by Page[11] in 1939. The diagnosis of arterial or, as it is more commonly called, essential hypertension, is therefore made by the exclusion of the other conditions which may present a similar picture.

Arterial hypertension is a highly complex disease syndrome characterized at first by only a mild elevation in the systolic

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and diastolic blood pressures. From then on its manifestations are many and the progress of the disease varies greatly. One patient may have more or less incapacitating symptoms of headache, nervousness, insomnia and ease of fatigue but without evidence of cardiac or renal damage. Another with apparently the same elevation in blood pressure may be practically symptom-free but will shortly begin to show evidences of cardiac disease. Still another patient may have the usual hypertensive symptoms and some evidences of cardiac involvement but on careful examination it is found that the kidneys are chiefly affected. Another patient may first manifest evidences of this disease by a so-called stroke, either a cerebral hemorrhage or a cerebral arterial thrombosis.

The rapidity of progress of the disease varies as much as its manifestations. A few live out a normal life span without any untoward incident but the majority sooner or later have serious constitutional changes with cardiac, renal or cerebral involvement. No one can definitely prophesy when such serious organic involvement may occur. Some patients will go with what appears to be an apparent benign form of hypertension for many years, even without the more common symptoms of the disease, and then suddenly and for no known reason develop rapidly severe changes in the heart or kidneys or suffer a cerebral hemorrhage. The more common course is for the disease to progress slowly, the symptoms gradually increasing in number and severity and within a few years cardiac, renal or cerebral involvement becomes manifest. A few patients present a fulminating type of the disease called malignant hypertension with death ensuing in a few months.

The manifestations of hypertension are so many and so varied and the actual progress of the disease so uncertain, embracing a course from the extreme of a fatal termination in a few months to a seemingly benign course for many years, that a true evaluation of any form of therapy is extremely difficult. This probably accounts for the very large number of therapeutic measures that have been advocated. Very thorough and honest appraisals have been made of all the usual drugs recommended for the treatment of hypertension and it has been unequivocably demonstrated that they are of no practical value.

It is true that a number of drugs will very temporarily reduce the blood pressure but such temporary reduction is of no value in the treatment of a chronic disease. Nitroglycerine is of course indicated in the emergency of angina pectoris but only for its immediate and extremely temporary effect. It is valueless in preventing future attacks. The only drug which today is recommended by some of our leading medical men is potassium thiocyanate. This must be given with a great deal of caution and frequent blood level determinations obtained. Even with the most careful precautions and when the level is maintained at a theoretical safe concentration, toxic symptoms sometime develop; and it is because of this uncertainty, both in its toxicity and in its therapeutic benefit, that many leading internists do not recommend it. It is therefore fortunate that surgery offers worth while improvement in a significant percentage of cases.

The cause of hypertension is unknown but an ever increasing mass of data indicates that heredity plays an important rôle. The vast majority of cases give a definite family history of hypertension. In fact it is rather unusual to get a history of a patient neither of whose parents had an abnormally high blood pressure. Commonly, in addition to either the father or the mother having had hypertension, we obtain a history that one or more brothers or sisters of that particular parent also had an elevated blood pressure. In some families there is a very definite history of several dying from cerebrovascular accidents. In another family we will have renal complications as the cause of death. In a few we

have been able to trace the hypertension for three generations. With a hypertensive family history we can be quite certain that one or more of the children will have hypertension and the percentage greatly increases if both parents are hypertensive.

Frequently the disease appears at a younger age in the children than in the parents. One young woman who came to us for a splanchnicectomy to relieve her hypertension gave the following striking family history: Her mother had died of cardiorenal complications from hypertension, her mother's sister likewise had hypertension and died of cardiorenal disease. The patient had five sisters, she being the youngest in the family; all five sisters had had hypertension and had died before the patient came to us. We have had numerous other family histories nearly as striking. Certainly with such evidence the rôle of heredity must be given very serious consideration. We do not believe that it is an abnormality in the autonomic nervous system which is inherited. This variation from the normal is manifested by excessive responses to normal stimuli. In other words, we believe that there is a congenital tendency of the autonomic system to overreact and to respond to a given stimulus by either greater or more prolonged vasoconstriction, which in the hypertensive involves the renal circulation.

The development of hypertension is usually insidious and many times an early hypertensive case is discovered largely by accident. The routine physical examinations required by many industrial firms for all applicants at the time of employment frequently disclose some elevation in blood pressure in young individuals who are completely symptom-free. A surprisingly large number of early hypertensive cases was found during the induction examination for the Army and Navy. Life insurance examinations have been a fruitful source in the discovery of hypertension. In spite of these various opportunities for the detection of the disease the great majority of patients do not know that they have an abnormally elevated blood pressure until they consult a physician because of severe symptoms such as incapacitating headaches and abnormal nervousness and irritability, or because of actual organic changes in the heart or kidneys. It is therefore impossible in the large proportion of our patients to make a definite statement of how long hypertension has actually existed.

Certain precipitating factors seem at times to be the immediate cause of hypertension; particularly is this true of pregnancy. Rather commonly the history discloses that hypertension first developed during pregnancy. Sometimes the blood pressure drops to normal after delivery at full term only to rise again and generally to a higher level at a subsequent pregnancy. In such cases the blood pressure level seldom comes down after termination of that pregnancy. We are of the opinion that the pregnancy simply precipitates the disease in a woman who, in all probability, would have developed hypertension at some time later. In other words she was a potential hypertensive individual and it took only this added change in her physiology to bring on the hypertension at an earlier date.

Unfortunately, it is impossible to prove or disprove this assertion. The toxemia of pregnancy appears to be much more common in women who do have elevated blood pressure and it has been shown that roughly 25 per cent of women who have had such a toxemia have a persistent hypertension postpartum.[3] The magnitude of this factor in the over-all picture of hypertension is shown by the following figures: toxemia occurs in about 8 per cent of all pregnancies, therefore, 2 per cent of all women who become pregnant will be expected to develop a hypertension which persists.[19] At the present rate of births in this country this would give a figure of more than 70,000 women each year who can be expected to develop hypertension as the result of pregnancy.[10]

Prolonged emotional strain such as worry over financial matters or domestic problems is frequently given as a precipitating cause of hypertension. Certainly in times of severe stress, as in war, financial crises or some other widespread causes for severe anxiety, the number of hypertensive patients seems to be greatly increased. There is also considerable evidence that very severe illnesses, such as pneumonia, mastoiditis and often such short illnesses as acute appendicitis, may be precipitating factors. However, such opinions are exceedingly difficult to prove since the majority of people do not have their blood pressures taken until real need for medical attention develops.

DIAGNOSIS

The diagnosis of arterial hypertension, commonly spoken of as essential hypertension and by some as diastolic hypertension, is primarily based on the findings of an elevated systolic and diastolic pressure. A single observation is of little significance. Repeated pressures must be taken under various circumstances. Several criteria to determine the hypertensive state are in use. Goldring and Chasis[5] make the following statement, "From three to sixteen years of age the systolic blood pressure rises progressively from 90 mm. Hg to 115 mm. Hg and the diastolic from 65 mm. Hg to 75 mm. Hg; at forty years of age the upper limit is 140 mm. Hg in the systolic and 90 mm. Hg in the diastolic phase; above forty the upper normal limit is 150 mm. Hg in the systolic and 90 mm. Hg in the diastolic phase." Some authors are more extreme and consider a diastolic reading of over 80 mm. abnormal at any age. However, the majority of medical men consider the diastolic as normal if below 100 mm. at age forty or older. The method of taking the blood pressure accounts for many discrepancies. Usually the blood pressure is different in the reclining, sitting and standing positions. It may differ considerably in the two arms.

The emotional status of the patient at the time of examination is an important factor and must be considered in each case. A patient manifesting nervousness over the examination or anxiety over some other matter will have a pressure definitely above his usual level. I saw this demonstrated many times during the war when young men who were particularly desirous of getting into the airforce were refused because of a slightly elevated blood pressure. On numerous examinations, their blood pressures were found to be absolutely normal by their home physicians and by myself. Almost routinely the blood pressure will be found at a lower level, both in normal and in hypertensive patients, if the reading is taken following a short rest instead of being taken immediately upon entering the examining room. It is believed by some that a pre-hypertensive stage can be demonstrated, but there is much conflicting evidence in this matter and at present we have no test which will definitely indicate whether a certain individual will or will not subsequently develop hypertension. However, certain leads have been established and it can be stated quite definitely that the individuals who maintain a relatively stable normal blood pressure level under a variety of conditions and at different times will probably not develop hypertension. Only about 3 per cent of such apparently stable individuals have subsequently been found to have hypertension. Conversely, 70 per cent of apparently normal individuals who showed temporary elevations in systolic and diastolic pressures at various times subsequently developed hypertension.

The blood pressure should be taken initially in the horizontal, sitting and standing positions and in each arm. If an abnormally high level is recorded, the patient should rest for at least fifteen minutes, preferably longer, in a horizontal position and the blood pressure readings again recorded. Useful information is sometimes obtained by having this type of patient exercise such as running up and down a flight of stairs, recording the blood pressure

changes from such a standard type of exertion. As a matter of routine we always take three blood pressure readings in quick succession in each arm in each position of the body. If considerable variation is found on these three readings, subsequent readings are taken after a few minutes. It is possible to take direct arterial pressures by using a needle placed into the lumen of an artery, but for all practical purposes the mercury manometer or an accurate aneroid manometer with auscultatory determination of the pulse sounds at the elbow is satisfactory. It is desirable in all young people to determine the pulse pressure in the femoral artery since this will determine the presence or absence of coarctation of the aorta as a possible cause of the hypertension.

SYMPTOMS

Certain symptoms are so regularly complained of by hypertensive patients that one can be almost certain of the diagnosis, in many cases, before having any knowledge of the actual blood pressure level. However, in the very early case, symptoms are usually absent; even in advanced hypertension, symptoms may be entirely wanting. There does not seem to be any correlation between the height of blood pressure and symptoms. Some patients with a systolic blood pressure of 190 will complain of excrutiating suboccipital headaches; other patients with pressures ranging from 240 to 260 or even higher may never have a headache. However, it is generally noted in patients who do have symptoms that these are aggravated when the pressure is at an unusually high level.

The common symptoms of which a large number of patients complain are: headaches, especially suboccipital, nervousness, irritability, insomnia, nocturia, shortness of breath and ease of fatigue. The headaches are extremely common on awakening in the morning and disappear after the patient is up and about. In the more advanced cases the headaches may awaken the patient very early, sometimes at three or four in the morning, and it is impossible for them to get back to sleep even after taking the usual headache remedies. The headache may be bifrontal or bitemporal or extend over the entire head but most commonly it is in the back of the neck and suboccipital region and from there at times extends forward to the top of the head and frontal area. Severe suboccipital headaches occurring more or less regularly every morning are so characteristic of hypertension that when such a history is elicited I always expect to find a very material elevation in blood pressure. Some patients have a history of such headaches for many weeks at a time; others will complain of having them only one or two mornings a week. There may be periods, especially when the patient is on vacation or not under stress, when the headaches may entirely disappear. Frequently the taking of food and moving about will give relief but in some cases the headaches persist throughout the day. They may also appear at any time during the day especially if the patient becomes excited or is under stress or physically fatigued. It is rather a common medical practice to advise a patient to lie down in the afternoon and rest but many patients will state to do so will again bring on a suboccipital headache. At times these headaches are so severe as to be completely incapacitating. Generally there is no nausea or vomiting associated with them. Some patients will have complete relief of early morning headaches by sleeping with the head of the bed elevated. This is most easily accomplished by putting blocks of wood under the head of the bed raising it to a height of about 12 inches. In extreme cases the patient refuses to go to bed and sleeps sitting up in a chair.

Increasing nervousness is an exceedingly common complaint although some patients are not aware of this themselves and the family have to give the definite history. Not infrequently patients will state that they have always been rather nervous but never to the extent that they have felt since the onset of hypertension.

Frequently associated with increasing nervousness is irritability. This may be a comparatively minor complaint but in many patients it assumes really serious proportions. It may become so pronounced as to interfere with his business and with his domestic happiness. I have frequently had a patient make the following statement to me, "I love my children as much as any father possibly could, but it has gotten so now that anything they do, no matter how trivial, irritates me seriously." The nervousness and irritability are not dependent upon the height of the blood pressure. Some patients having this complaint have only moderately elevated pressures, while others who may have a very marked hypertension are relatively calm. However, in the majority of cases the nervousness and irritability are greater during periods in which the pressure is unusually high. A vicious circle certainly exists, the increased pressure making the patient more nervous and more irritable and this conversely raising the pressure still higher.

Nearly all patients complain of nocturia, generally once or twice at night, but in an appreciable number of cases nocturia is a very real problem and seriously interferes with sleep. A history of having to urinate every hour during the night is not especially uncommon. This is particularly found in those individuals whose concentrating ability is seriously impaired. Nocturnal dyspnea fortunately is a rather uncommon symptom since it indicates very serious cardiac damage. Very mild nocturnal dyspnea may be completely relieved by sleeping with the head on several pillows, but in the more severe cases the patient is unable to sleep in bed and has to sit in a nearly upright position in a chair

Weight reduction, if the patient is obese, and digtalis may be of great value in relieving this distressing symptom but the over-all picture will always remain grave. Ease of fatigue is an exceedingly common complaint in hypertensive patients. It may be due somewhat to increased nervousness but seems more related to so-called vital capacity. It does not have a definite relationship with the height of the blood pressure but may have a relationship with the cardiac and renal status. Some patients present just the opposite picture and with the development of hypertension become more and more dynamic and tireless. They feel keyed-up and under tension and do everything quickly. This is the type of personality so commonly considered hypertensive. The complaint of feeling always keyed-up and under tension is not confined, however, to this group and is, in fact, an exceedingly common complaint of all hypertensive patients. They frequently state that they cannot relax and this in itself makes sleep difficult.

Shortness of breath on mild physical exertion such as going up one flight of stairs is an extremely common complaint. It may or may not indicate cardiac damage. However, if the symptom is very definitely progressive and the patient is not unduly overweight, cardiac disturbance can be expected. Palpitation, either under exertion or while quiet, is also a very common complaint. It does not necessarily signify any organic change in the heart. In fact it is much more frequently associated with nervousness. Angina pectoris, either in a mild or severe form, occurs commonly in hypertensive patients. It may be the result of simple coronary spasm but in many it is probably due to actual arteriosclerosis of the coronary vessels. Particularly is this true when the hypertension is of long duration. Patients with angina pectoris are advised to stop smoking and to limit their activities and emotional strain. Nitroglycerine is invaluable as a relief agent at the onset of an attack. The patient with frequent anginal attacks has certainly a much graver prognosis. Fortunately many patients with angina have complete relief following splanchnicectomy. This suggests that vascular spasm rather than arteriosclerosis was the immediate factor. Edema of the ankles when occurring regularly is of serious import since it is most frequently associated

with cardiac decompensation. Other causes such as varicose veins should of course be ruled out.

Nausea and vomiting are uncommon symptoms of hypertension even in the patients who suffer from severe headaches. A history of repeated attacks of nausea and vomiting is highly suggestive of the so-called malignant type of hypertension. In most patients with these symptoms evidences of severe renal damage will be found.

Mental symptoms of a more or less severe degree occur very commonly in hypertensive patients. As a rule they are slight and characterized chiefly by difficulty in concentration and a slightly impaired memory, especially for recent matters of little consequence. However, many patients complain that "their head feels thick," that they cannot think as they formerly did, that at times they seem to be in a fog; some complain that their loss of memory is a serious handicap in their business. Some patients have apparently not been cognizant of any previous mental difficulty until after a splanchnicectomy; then they commonly remark that they can now think clearly, that they can concentrate and they often state, "it seems as though a fog had been wiped away." From these mild evidences of hypertensive encephalopathy we have various grades in the individual who is entirely incapable of carrying on any active mental processes. Usually the mental changes are insidious and their development very gradual. Even marked mental deficiency may be found in patients who apparently have not had a cerebral accident, either cerebral hemorrhage or thrombosis. All the signs and symptoms of hypertensive encephalopathy are greatly increased when a so-called cerebral accident occurs. Small intracranial hemorrhages can occur without danger to life but I believe the majority of so-called strokes which the patient survives are actually due to vascular thrombosis. I do not deny the possibility of cerebral vascular spasm but believe, at least in the majority of cases, that even the transitory symptoms of a stroke are due to small thrombii.

Blurring of the vision, either recurrent and transitory, or of more prolonged periods is a rather common symptom in the more advanced stages of hypertensive disease. At times the blurring lasts only for a few minutes and strongly suggests angiospasm. More prolonged periods of blurring in one eye or in both are most commonly associated with retinal hemorrhages. As a rule such hemorrhages are associated with a rather marked hypertension but may occur in individuals whose pressure is not over 170 systolic. It is also true that in some individuals who have exceedingly high blood pressures, the fundoscopic examination never discloses a hemorrhage. Sudden complete loss of vision in one eye generally indicates either a massive retinal hemorrhage or a thrombosis of a central artery or vein or one of their main branches. A massive retinal hemorrhage may be slowly absorbed and useful vision finally restored, but a thrombosis of the central artery results in complete, permanent loss of vision. Impaired vision which fluctuates to some extent may be due to repeated small retinal hemorrhages and exudates but as a rule these do not impinge upon the macula and rather extensive hemorrhages are often seen without noticeable impairment of vision. Severe bilateral impairment of vision is most often due to a high degree of papilledema with the usually associated extensive hemorrhages and exudates. The finding of papilledema indicates a fulminating type of hypertension usually designated as malignant hypertension. The progress of the disease in these cases is so rapid and the prognosis so poor that many consider malignant hypertension as a separate disease entity.

Other hypertensive symptoms rather commonly complained of but which are not indicative of any disease picture are dizziness, vertigo, tinnitus and mild deafness. Many hypertensive patients com-

plain of transitory dizziness, sometimes associated with severe suboccipital headaches, at other times only present upon sudden changing of position. True vertigo is quite rare. Many patients complain of tinnitus, generally in both ears, and of a rather mild degree. Much more frequently patients complain of feeling the heart beat in the ear especially when reclining. Mild deafness certainly can be on a hypertensive basis since careful audiometer examinations prior and subsequent to splanchnicectomy have shown definite improvement in the hearing following operation.

TREATMENT

The commonly accepted drugs for the treatment of hypertension have been proved valueless. Certain general measures, however, are indicated, especially in the early or mild hypertensive patients. Reduction in weight is important in all obese patients. Occasionally reduction to a normal weight will materially lower the hypertension. In all cases it takes an added strain from the heart. When surgical therapy is advised, weight reduction is definitely indicated since the operation is much easier of accomplishment and the patient's subsequent care and comfort is enhanced. Of course, not all patients with hypertension are overweight; some in fact are below normal. In such individuals supplementary feedings, especially of a rather high caloric diet, are indicated; and if the appetite is poor, multivitamins may do much to improve it. Adequate rest at night is important and whenever possible a rest period after the noon meal should be taken. Few patients can afford to follow the advice so commonly given, namely, to give up all their work and to stop worrying about their condition. Worry is a recognized factor in heightening the blood pressure, but it is an impossibility for the average person to have to give up a gainful occupation and not worry about doing so.

Many special diets for the treatment of hypertension have been recommended but with two exceptions they are probably of little value. The exceptions are the rice diet[8] and the very low sodium diet.[1] Some patients adherring strictly to the rice diet have shown rather remarkable lowering of blood pressure and amelioration of their symptoms. However, on returning to a regular diet the evidences of hypertension quite promptly return. A low sodium diet, that is one containing only 200 mg. of sodium per day, has been found equally efficacious; some patients have a reduction in symptoms and in a few cases some reduction in blood pressure. If kidney function is adequate, radical reduction in protein intake is not indicated. However, if the blood non-protein nitrogen is rather markedly elevated, the diet should be low in protein. For weight reduction we generally recommend a 1,200 calorie diet; but in very obese patients or in those in whom weight reduction preparatory to a greatly needed operation must be accomplished rapidly, we have prescribed a diet of only 800 calories, supplementing this, of course, with vitamins. On a few occasions a diet as low as 350 calories has been prescribed.

Potassium thiocyanate has been highly recommended by many clinicians. It must be given with caution and the blood level determined at weekly intervals. It is generally considered that a level of at least 6 mg. per cent is necessary to have any therapeutic value and the level should not go above 12 mg. per cent. However, some patients show severe toxic reactions at levels even below 6 mg. per cent and for this reason many clinicians refuse to use this drug. It is a palliative measure at best. The symptoms may be alleviated or actually disappear and in some cases the blood pressure shows satisfactory reduction, but as soon as the drug is discontinued the hypertensive symptoms and elevated blood pressure return.

We believe that surgery offers the best form of therapy and a sufficient number of patients have now been operated upon and studied for a sufficiently long period

to make definite statements as to the value of splanchnicectomy. We do not advocate surgery for all patients. A very mild hypertensive patient may live for many years without apparent organic damage. However, if there are evidences of a progression of the disease, surgery seems definitely indicated. Far too often the medical man watches the progress of the disease until irreparable damage has resulted, either in the brain, the heart or the kidneys and then refers the patient for surgery. This is neither fair to the paitient, the surgeon nor to the procedure which he advocates.

RATIONALE FOR SURGICAL TREATMENT

The rationale for the surgical treatment of hypertension is based upon our concept of the disease, particularly of its etiology. Although many theories have been advanced, no one yet has actually been able to establish the true, primary cause of arterial hypertension. We have accepted the renal-humeral theory, first satisfactorily demonstrated by Goldblatt,[4] who showed conclusively that renal ischemia, or at least the interference with the renal hemodynamic state, produced a continuous elevation of blood pressure without at first any evidence of impaired renal excretion. It has been shown by numerous workers that such interference with the blood supply of the kidneys results in an excessive amount of a renal pseudoglobulin called rennin being liberated into the renal veins. This rennin reacting with another pseudoglobulin probably arising in the liver and named preangitonin (renninactivator of Page) forms an active vasopressor substance called angiotonin. This latter substance causes arteriolar constriction of sufficient degree to raise peripheral resistance throughout the body with a consequent elevation in blood pressure. Goldring and Chasis[5] make the following statement, "Hypertension is the sign representing the alteration in hemodynamics, and hypertensive disease is a clinical entity in which an unknown pressor mechanism initiates arteriolar vaso-constriction, elevated blood pressure, and vascular sequelae." We believe this vasopressor substance is angiotonin. The question naturally arises, What is the cause of this change in renal hemodynamics? There are probably numerous causes but we believe in the majority of cases it is of neurogenic origin.

The recent brilliant experimental work of Trueta, Barclay, Franklin, Daniel and Prichard[4] has clearly demonstrated that the splanchnic nerves can be reflexly stimulated with resulting severe renal ischemia. They have shown, for example, that stimulation of the sciatic nerve can reflexly stimulate the splanchnic nerves with a resulting vasoconstriction in the arterioles of the renal cortex so that the blood flow is partially or even completely abolished through this portion of the kidney. Direct stimulation of the distal end of the cut splanchnic nerve gave the same results. It had previously been shown that electrical stimulation of the splanchnic nerves in dogs elevated the blood pressure, probably by constricting the arterioles in the kidney, resulting in renal ischemia.

It is our opinion that in human hypertension an abnormal stimulation of the splanchnic nerves originates in the autonomic centers of the brain, resulting in similar changes in the renal hemodynamics. The activating stimulus may be psychic, reflexes from stimuli originating in other parts of the body, or it may be chemical in nature. Normally stimuli are constantly influencing the sympathetic nervous system but in certain diseases these stimuli result in excessive responses. This is seen for example in Raynaud's disease. We believe that the abnormal excessive response to normal stimuli, sufficient to cause renal ischemia, is based on a congenital abnormality in the autonomic nervous system whereby excessive responses are evoked by the normal stimuli reaching those centers. It is our opinnion that this tendency of the autonomic system to overreact, to respond to a given stimulus by either greater or more pro-

longed vasoconstriction of the renal vessels, is inherited. The family history of most hypertensive patients lends weight to this theory. If our theory is correct and there is a large amount of evidence to support it, splanchnicectomy if performed before permanent changes occur in the blood vessels should improve the renal hemodynamic state.

SELECTION OF PATIENTS FOR SPLANCHNICECTOMY

We believe that any patient showing a progressive type of hypertension and whose age and cardiac and renal status is satisfactory should have the benefit of surgical intervention. Age certainly is a factor. The younger group have a much higher percentage of worth while results than the older age groups. However, so many factors determine the ultimate result of the operation that each individual case should be considered on its own merits. We have more or less arbitrarily, fixed an age limit of fifty-three. When we originated the procedure of bilateral supradiaphragmatic splanchnicectomy for the treatment of hypertension fourteen years ago, we set an arbitrary age limit of forty-five; but it was soon found that a rather high percentage of patients at that age frequently responded very well and so the age limit was gradually extended, still with gratifying results. We may still further extend it. At present we seldom operate on a patient age fifty-four or above, unless that patient has incapacitating symptoms or shows evidence of a very rapidly progressive type of hypertension such as the so-called malignant phase. The youngest patient we have operated upon was aged eight, the oldest sixty-three.

The question frequently arises, Should a patient who has had a cerebral accident be operated upon for the treatment of his hypertension? We realize that a cerebral accident presupposes abnormal pathologic changes in the cerebral vessels and that the risk and the prognosis of such a patient is influenced thereby. Under no circumstances would we operate upon a patient shortly after a cerebral hemorrhage or thrombosis; but if the patient has made a complete recovery both physically and mentally and in addition shows a progressive type of hypertension, we would advise operation. However, if there is evidence that the blood pressure has been remaining at the same level for a prolonged period and the patient has had no increase in symptoms or further impairement of renal or cardiac condition and especially if the patient is over fifty, we would probably advise against splanchnicectomy.

Cardiac decompensation is, of course, an absolute contraindication. However, some patients who have given a history of noctural dyspnea, ankle edema and excessive shortness of breath on mild exertion but who have responded satisfactorily to digitalis and bed rest may be operated upon with a comparatively low operative mortality and definite hopes of at least temporary improvement. It should be pointed out that the majority of deaths occurring in the first five years postoperatively have been among those patients who had had such evidences of cardiac decompensation before operation.[5] Deaths within this period have occurred in patients whose blood pressure had been reduced and maintained at a normal level up to the time of death, two, three or more years following operation.

Renal insufficiency as measured by the level of the blood non-protein nitrogen and blood urea is also a definite contraindication to operation. We have found that only in exceptional cases have the patients had more than temporary improvement if the blood non-protein nitrogen, when the patient had been on a large fluid intake, was above 45 mg. per cent. Such temporary improvement might be for a year or more and possibly could be considered a justification for the operation. However, we generally do not advise operation if the non-protein nitrogen is above 45 with the single exception of those patients with malignant hypertension. There we realize that a defi-

nite emergency exists and we are willing to operate when the non-protein nitrogen cannot be brought below 56 mg. per cent.

The duration of hypertension probably is a factor in the ultimate outcome but unfortunately it is only an exceptional case in which the actual duration is known. We therefore do not have sufficient data upon which to base a positive statement. The various tests such as intravenous pentothal, sodium amytal, spinal anesthesia, cold pressor tests and so forth, which have been used so extensively in the hopes of finding some criteria which would definitely indicate the probable result of the operation, have proved valueless. It is true that some of these tests may show a general trend which statistically is of interest, but in the determination of the probable outcome of any individual they need not be considered.

Malignant hypertension is a definite indication for splanchnicectomy rather than a contraindication as some believe. It is the only form of therapy offering any chance for improvement in this type of hypertension. The prognosis is so absolutely hopeless without operation and the results of splanchnicectomy in some cases have been so brilliant that we advise operation even when the blood non-protein nitrogen is at a higher level than would be acceptable for the less serious case.

Certain tests are routinely made on all hypertensive patients since it is necessary to have full data on the cardiac and renal status before making a decision regarding a splanchnicectomy. The fundoscopic examination is also important since it gives us valuable information regarding the type of hypertension and its probable rate of progress. Full preoperative studies with similar postoperative studies over a period of years are essential if we are to evaluate properly the results of surgery.

The ophthalmologic examination is principally of the fundus although the visual acuity is recorded and in certain cases visual fields are made. The fundoscopic examination should be made with the pupil well dilated. The condition of the arteries and veins and the presence of angiospasm and the degree of arteriosclerosis is noted. The recognition of angiospasm as distinct from arteriosclerotic changes requires much practice. It is generally believed that the presence of angiospasm denotes a progressive type of hypertension. In the early stages of the disease the only pathological condition noted may be an increase in the reflex stripe of the arteries and arteriovenous nicking. Hemorrhages both striate and flame-shaped indicate a more serious phase of hypertension. The presence of cotton wool exudates, with or without associated hemorrhage, also indicates rather advanced hypertension. Edema of the borders of the discs should be carefully looked for and the actual presence of papilledema of 1 diopter or more is considered pathognomonic of malignant hypertension.

The cardiac status is determined by an electrocardiogram, an orthodiagram and a teleoroentgenogram. If there is evidence of coronary involvement, an additional electrocardiogram with chest leads is made. In some special studies ballistocardiographic records have also been made. The data from the above findings are of course correlated with the clinical history, with particular attention to shortness of breath on exertion, nocturnal dyspnea, ankle edema and attacks of angina pectoris.

The renal function is determined by complete urinalysis, urea clearance, water concentration, blood non-protein nitrogen and blood urea, and intravenous pyelograms. Formerly we used a thirty-six-hour water concentration test but this proved extremely burdensome for many of the patients and in recent years we have compromised by using an eighteen-hour test made in the following way. The patient has his usual meal at 6 P.M., then no food or fluids of any kind are taken until afternoon of the following day. A urine sample is obtained at 8 A.M. again at 10 A.M. and again at 12 noon, and the specific gravity and presence and amount, if present, of albumin separately determined. This test has given somewhat lower values than we

obtained on the thirty-six hour test. The blood non-protein nitrogen and blood urea determinations should be made when the patient is on a large fluid intake. Intravenous pyelograms have been made on all patients to rule out gross pathologic disorder in either kidney. Retrograde pyelograms are necessary when the intravenous pyelograms do not give unequivocal data. Considerable discussion has arisen over the requirement of an intravenous pyelogram since it is comparatively rare to find a gross pathologic condition sufficient to be a possible cause of the hypertension. Possibly it is only necessary to make this test in those individuals who give a suggestive history of severe renal complications and in children in whom a possible congenital renal lesion may be present. The presence of bilateral chronically contracted kidneys is a definite contraindication to operation, so too are polycystic kidneys. When one kidney is found by x-ray to be diseased and the other normal, a nephrectomy has been advised but in only a few cases has this materially improved the hypertension. In many cases the subsequent bilateral splanchnicectomy has given excellent results. Craig,[2] who performs his splanchnicectomies in two stages, advocates a unilateral splanchnicectomy on the side of the diseased kidney at the time of nephrectomy; then later if satisfactory results have not been obtained, the second stage operation for splanchnicectomy on the opposite side is performed.

The use of tetra-ethyl-ammonium as a test for operative prognosis has been made in a rather large series of cases. Like other tests involving generalized vasodilation, it has been found of no definite value in the prognosis of any single individual; however, certain generalities can be made.[9] In a large series it is demonstrated that those patients who respond with a significant drop in blood pressure with the administration of tetra-ethyl-ammonium intravenously will show a high percentage of good results following splanchnicectomy, and conversely the group who do not respond to tetra-ethyl-ammonium have a low percentage of satisfactory lowering of the blood pressure following surgery. However, the test in no way indicates whether the patient will receive worth while symptomatic relief. It has been repeatedly demonstrated that the relief of distressing symptoms is not closely correlated with the reduction of blood pressure. Tetra-ethyl-ammonium should not be used as a therapeutic agent but simply as a test of the response to a vaso-dilating drug to help determine the possible result of operation.

Many hypertensive patients show evidences of encephalopathies varying in degree from slight loss of memory or difficulty in concentrating to marked mental clouding. We are now making electroencephalograms using an 8 channel machine on all such patients. Similar recordings will be made at suitable postoperative periods. We hope with these data to be able to determine how much of the hypertensive encephalopathy is due to actual organic changes and how much improvement in the mental status, following splanchnicectomy, may be expected.

SURGICAL PROCEDURES

The surgical technic for the treatment of hypertension varies in different clinics, chiefly in the extent of the sympathetic denervation. Since our first operation[12] for hypertension in November, 1933, we have routinely carried out a bilateral supradiaphragmatic splanchnicectomy.[13] In some clinics,[8–12] this is combined with resection of the first or first and second lumbar ganglia subdiaphragmatically. Other variations in the procedure extend the excision of the thoracic ganglia up to include the third and in one clinic the first thoracic ganglion.[6]

The operation of bilateral supradiaphragmatic splanchnicectomy has now been performed in our clinic on about 2,000 patients over a fourteen-year–period. It consists of the bilateral resection of a very long segment of each greater splanchnic nerve and of the lower thoracic sympa-

thetic ganglia with the lesser and least splanchnic nerves arising from them. In our earlier cases we resected about 10 to 12 cm. of each greater splanchnic nerve and excised the tenth, eleventh and twelfth sympathetic ganglia bilaterally. With added experience, the addition of special instruments and a new form of lighted retractor we have been able to resect routinely 20 cm. or more of the greater splanchnic nerve and the seventh to the twelfth inclusive sympathetic ganglia. In some cases it is possible to resect the sixth and even the fifth thoracic ganglia through the same approach used formerly, namely, resection of a portion of the eleventh rib. Avertin anesthesia, using 110 mg. per Kg. of body weight, supplemented with nitrous oxide and oxygen, has been the anesthetic of choice. Intravenous physiologic glucose solution is given slowly throughout the operation.

Two scratch marks outlining the bilateral incisions are made, each about 10 cm. long on thin individuals, longer in heavily built or fat patients. These incisions are parallel in vertical direction, about 8 cm. apart and center over the eleventh intercostal spaces. The incision is carried through the skin, superficial fat and lumbodorsal fascia. The longissimus dorsi is retracted medially and the eleventh rib exposed. The intercostal muscles are separated from the rib as is the pleura and a segment 5 or 6 cm. long of the rib resected. The excision extends nearly to the head of the rib. A small piece of the intercostal muscle is removed and saved for biopsy examination to disclose pathologic changes in the small arterioles. The intercostal artery and vein lying just above the twelfth rib are double clipped and a segment about 5 cm. long excised. Care is taken not to damage the adjacent intercostal nerve. The pleura is then very carefully separated by semi-sharp dissection from the bodies of the vertebrae and this dissection is carried medially to the level of the anterior border of the vertebral bodies. The pleura is separated caudally to the crus of the diaphragm and cephalad as far as it can be conveniently exposed, usually to a level above the seventh thoracic ganglion. In some individuals the configuration of the chest is such that this dissection can be carried up above the fifth thoracic vertebra without difficulty. On the right side the greater splanchnic nerve lies between the pleura and the inferior border of the vertebrae; on the left it lies on top of the aorta which in most hypertensive cases is displaced laterally. The greater splanchnic nerve is grasped with long forceps and upward traction made. Frequently such traction pulls the upper pole of the celiac ganglion through the opening in the diaphragm for the passage of the greater splanchnic nerve. The nerve is then dessicated with an electric current as it enters the ganglion and divided. The distal stump retracts below the diaphragm. The nerve is then carefully dissected from its bed of fascia and freed upward to the level of the sixth or seventh vertebra.

The thoracic ganglionated chain lies more dorsally on the bodies of the vertebra. The ramus from the eleventh intercostal nerve to the eleventh thoracic ganglion is dessicated, divided, and then the chain dissected downward exposing the twelfth ramus and ganglion. In many cases, because of rather high reflection of the diaphragm, it is necessary to divide some of the fibers of the diaphragm at their attachment to the vertebrae until the twelfth ganglion is fully exposed. This may even lie on the peritoneal side of the diaphragm. The chain below the twelfth is dessicated and divided and the least splanchnic nerve avulsed as the sympathetic chain is pulled upward. Resection is then carried upward with a special semi-sharp elevator and in turn the rami to the tenth, ninth, eighth, seventh and some times the sixth, rarely the fifth, ganglia are dessicated and cut and then the chain above the uppermost exposed ganglia is dessicated with the electric current and divided. As a rule the main trunk of the greater splanchnic nerve will be found arising from the uppermost of the

exposed thoracic ganglia. The entire chain and the attached greater splanchnic nerve are removed from the extrapleural space. The various lesser splanchnic nerves are either avulsed or, if they are especially strong, dessicated and divided at the level of the diaphragm. All bleeding points are carefully dessicated. The extrapleural space is then filled with Ringer's solution and the lung fully expanded by increasing the pressure in the rebreathing bag of the anesthetic machine.

If a hole is inadvertently torn in the pleura during any stage of the operation, it in no way modifies the procedure and the operation is continued without difficulty. If the pleura has been accidentally torn on the right side, we do not hesitate to proceed with the left splanchnicectomy. Of course special care is taken to assure full lung expansion before closure of the incision. Rarely is there suction into the pleural cavity during closure and in such cases aspiration through a needle at the completion of operation may be necessary. The reason for filling the extrapleural space with Ringer's solution before expanding the lung is to assure no air being trapped in the mediastinum which would of itself cause no serious complication but may result in some crepitation in the neck and be a cause of worry to the patient. Any Ringer's solution not forced out by the expansion of the lung is of course readily absorbed. The incision is closed in layers and immediately a similar procedure carried out on the left side. The combined bilateral operation takes from forty-five minutes to one and one-half hours. Frequent blood pressure recordings are made throughout the operation. If the blood pressure drops below 120 mm. Hg systolic, supportive measures are used. We have found neosynephrin very useful in maintaining the blood pressure at a safe level.

The blood pressure may drop at any time during the next few hours and therefore the patient is kept flat in bed with the foot of the bed elevated for the first twelve hours. It may be necessary to give neosynephrin on one or two occasions during the first few hours. Patients are encouraged to sit up at an early date and those who feel able to be out of bed may get up within a day or two following operation. On first assuming the upright position there may be a postural hypotension. This as a rule corrects itself within a few days. It has never been necessary to bandage the legs or put compression upon the abdomen. However, we always advise the patients first to assume the upright position for a few minutes, then to get out of bed slowly. Most patients leave the hospital on the twelfth postoperative day and it is rather unusual for patients to have to remain in more than fourteen days. We advise a rest period at home of at least four weeks before resuming any active duties.

Few complications have arisen in about 2,000 operations. Five patients have had an injury to the thoracic duct and have developed extrapleural collections of chyle. Three of these were successfully treated by repeated aspiration for a few days. The other two required operation; in one the leaking thoracic duct was found damaged at the crus of the diaphragm; in the other a small radical lying on top of and to the outer side of the aorta about the level of the tenth interspace had apparently been torn. A clip upon this radical stopped further leakage of chyle and convalescence was uninterrupted.

Occasionally a mild atelectatic condition has developed. Usually this has been controlled by insisting upon the patient coughing but on a few occasions it has been necessary to bronchoscope the patient and aspirate the mucous plug. Pneumonia has not been a complication for several years. Previously, when atelectasis had not been recognized, a few patients developed mild bronchial pneumonia. Because we insist upon the patient moving about freely and encourage early ambulation, thrombophlebitis of the lower extremities has not occurred.

A few cases of coronary thrombosis, either during the operation, immediately

after or before discharge from the hospital, have occurred, but in none who did not have a previous history of coronary disease. A few individuals have had cerebral thrombosis, apparently as a result of a marked drop in blood pressure occurring during or immediately after the operation. These were all in individuals with considerable evidence of cerebral arteriosclerosis. It is for this reason that we are exceedingly careful to keep up the blood pressure to at least 120 mm. Hg systolic throughout the operation and during early convalescence.

SUMMARY OF INDICATIONS AND CONTRAINDICATIONS FOR SPLANCHNICECTOMY

Our criteria for operation may be summarized as follows: an age below fifty-four but with occasional exceptions; a more or less continuously elevated blood pressure with a systolic over 170 and a diastolic over 105; definite evidence in the milder hypertensive cases of a progression in the disease picture; a non-protein nitrogen below 45 mg. per cent, preferably below 40 mg. per cent; a compensated heart without recent history of coronary thrombosis. We prefer that the patient have a relatively normal cerebral function but have operated upon many showing rather marked hypertensive encephalopathy with gratifying results. Exceptions to the above general rules have been made, especially in operating upon patients who are older, generally because of incapacitating symptoms or evidences of a rapidly progressive type of hypertension. Only in the malignant type do we believe operation is worth while if the non-protein nitrogen is above 45 mg. per cent.

CLASSIFICATION OF HYPERTENSION

The manifestations of hypertension are so varied that it is necessary to have a workable classification in order to evaluate properly the results of surgical therapy. Even with a very complex classification so many factors are involved that a true statistical study is difficult. Arterial hypertension is a generalized disease, all organs of the body being affected, although the principal pathologic disorder may be greatest in one or two important organs. The commonly accepted classification is that of Wagener and Keith[22] but we did not find this entirely satisfactory and hence Isberg and I[15] enlarged the grouping from four to six. We suggest the following classification as very useful since it emphasizes the dominant pathologic picture:

Group 1. Early, mild hypertension. These patients are entirely asymptomatic, have normal or grade 1 fundi, and show no evidence of cardiac, renal or cerebral involvement.

Group 2. Symptoms predominate. All patients in this group complain of symptoms and have mild changes in the retinal blood vessels but display no evidences of cardiac, renal or cerebral impairment.

Group 3. Organic heart disease is predominant. In each case the diagnosis of heart disease is confirmed by either or both a definitely abnormal electrocardiogram and a teleoroentgenogram showing cardiac enlargement.

Group 4. Cerebrovascular disease is predominant. Each patient in this group has had one or more previous cerebral accidents.

Group 5. Impaired renal function is predominant. Each patient shows diminished concentrating ability and urea clearance values.

Group 6. Malignant hypertension. These patients have severe neuroretinitis with definite papilledema of one diopter or more and display a rapidly progressive, downhill course.

RESULTS OF BILATERAL SUPRADIAPHRAGMATIC SPLANCHNICECTOMY FOR HYPERTENSION

A bilateral supradiaphragmatic splanchnicectomy has been performed upon approximately 2,000 patients at the University Hospital over a period of fourteen years. A large proportion of these patients have

returned to the hospital for periodic examinations. Some have returned yearly; others only after a period of several years. Very complete postoperative studies have been made by the home physician in many cases when the patient could not come to us. We have attempted to obtain in each case complete postoperative studies, including a careful interval history, ocular fundoscopic examinations, water concentration, urea clearance, blood nonprotein nitrogen, electrocardiogram, orthodiagram and repeated blood pressure readings. Unfortunately it has not been possible to get complete studies on all the patients returning for check-up examinations. This accounts for the different numbers of patients included in the statistical studies on postoperative blood pressure levels and renal and cardiac status. We believe an interval of at least one year following operation should elapse before making any evaluation of the results of the procedure. Some of our statistics are based on studies from one to nearly fourteen years following operation. Special long term studies have been made at one postoperative period five to thirteen years.

SYMPTOMATIC RELIEF

This has been striking and in general is apparently only roughly correlated with the reduction in blood pressure. However, certain symptoms such as shortness of breath and nocturia are quite closely related with improvement in cardiac and renal function. The excruciating headaches which are usually suboccipital and the marked nervousness and irritability are in most cases completely relieved. Insomnia, nocturia and distressing palpitation of the heart have been completely or at least greatly improved in 86 per cent of the patients still living. This percentage of improvement has persisted almost unchanged for postoperative periods of from five to twelve years. Visual improvement has been striking especially in the malignant cases in which vision is often very seriously impaired. In every surviving patient who had had visual difficulties on a hypertensive basis the improvement has been practically 100 per cent. Some who were so blind that they could not distinguish faces have had a return to normal visual acuity, usually within a very few weeks.

BLOOD PRESSURE RESPONSE TO SPLANCHNICECTOMY

Blood pressure, both systolic and diastolic, was significantly reduced over a period of five to twelve years after operation in 81.3 per cent of the patients still living.[15] No improvement in blood pressure level was found in 12.7 per cent but over the course of these years only 6 per cent had had an increase in blood pressure. If we include in this statistical study those patients who had died one to twelve years postoperatively with the group still living five to twelve years after operation, we have definite improvement in the blood pressure in 46.7 per cent of the entire series. It should be borne in mind that 49 per cent of those that died in this period had malignant hypertension. We have used the following criteria for normal blood pressure readings: age twenty to forty years—140/90; age forty to sixty years—150/95. While some disagree, these figures are generally accepted. Using this standard 20.3 per cent of our patients have maintained normal blood pressures following splanchnicectomy for five to twelve years. Those showing a worth while reduction to normal or approximately normal at the end of one year have, with very few exceptions, maintained the same blood pressure level at each subsequent examination, sometimes yearly, up to the longest postoperative period in our series, which is nearly fourteen years. A few cases have shown a return of the hypertension after maintaining normal levels from three to ten years postoperatively. It has been suggested that this signifies a regeneration of autonomic fibers. A marked improvement, but not to a normal level, based on a reduction of over 80 mm. Hg systolic

and 25 mm. Hg diastolic, has been maintained in 26 per cent of our patients for the entire postoperative period of five to twelve years. An additional 35 per cent have had what we call significant improvement, that is a maintained reduction in blood pressure of more than 40 mm. systolic and 15 diastolic. Combining the three groups, those with a reduction to normal, those with marked reduction, and those with significant reductions, we have an over-all improvement in blood pressure in 81.3 per cent of the living cases, which have been maintained from five to twelve years. If we consider the blood pressure of those who died before the end of this period, most of whom were malignant cases, we still have an improvement in blood pressure being maintained in nearly 50 per cent of the cases.

FUNDOSCOPIC CHANGES AFTER SPLANCHNICECTOMY

Of our patients showing preoperative fundoscopic findings of angiospastic retinitis with or without hemorrhages or exudates 82 per cent showed no evidence of angiospasm, hemorrhages or exudates at any follow-up examinations five to twelve years after operation. Papilledema usually disappeared quickly and in the twenty-one cases of malignant hypertension still living five to twelve years after operation it had not returned.

HEART CHANGES AFTER SPLANCHNICECTOMY

The response of the hypertensive heart to splanchnicectomy has been very carefully studied.[16] Three hundred eighty-four patients who had been operated upon from five to twelve years previously and who had had complete studies, including electrocardiogram, orthodiagram and teleoroentgenogram, both before and at various periods after operation, showed the following results: It was gratifying that 60 per cent of the patients with known hypertensive heart disease were still living five to twelve years after splanchnicectomy and that 93 per cent of those who had normal hearts prior to operation were still living. Those showing abnormal preoperative electrocardiograms had significant improvement in the tracings in 41 per cent, five years or more after operation. Many patients with gross enlargement of the heart preoperatively have had return to normal size. A significant decrease in heart size has been maintained in those with definite preoperative cardiac enlargement over a five to twelve-year period in 44 per cent. Only 10 per cent of those with preoperative cardiac enlargement showed any further enlargement over the long postoperative period. Nearly all patients who have shown improvement in the electrocardiogram or decrease in heart size have maintained a significant decrease in blood pressure. As previously pointed out patients who had had congestive heart failure but who had responded to digitalis preparatory to a splanchnicectomy had a much poorer ultimate prognosis. Our recent studies have shown that only one in three of such patients will survive for a postoperative period of five to twelve years. Only 12.5 per cent of patients who had had frequent paroxysms of nocturnal dyspnea survived for this long period.

A hypertensive patient who has had a coronary occlusion with a myocardial infarction and whose blood pressure still persists at a high level presents a very serious prognosis. However, twelve such persons have been treated by splanchnicectomy without a fatality and nine of these were still living five to ten years postoperatively. This prolonged survival of 75 per cent of such severe cases is very encouraging.

Of the hypertensive patients with normal heart size prior to operation 86.6 per cent were still living and of these 92.5 per cent had maintained normal heart size.

RENAL CHANGES AFTER SPLANCHNICECTOMY

The renal functions show definite improvement in many cases following splanch-

nicectomy. Nocturia is almost completely relieved and 45 per cent of patients with abnormal urea clearance have returned to normal and 44 per cent with impaired water concentration have marked improvement. In the majority of cases albumin has completely disappeared from the urine.

PREVENTION OF CEREBRAL ACCIDENTS BY SPLANCHNICECTOMY

Excluding cases of malignant hypertension fifty-eight patients are still living five to thirteen years after operation of a total of 108 who had had definite cerebral accidents prior to operation. Fifty of the fifty-eight have had no subsequent cerebral insults throughout this long postoperative period. Ten hypertensive patients who had suffered strokes prior to operation have maintained normal blood pressure levels since splanchnicectomy, the shortest period being five years. These figures indicate that, even though the patient has had a cerebrovascular accident, surgery offers real hopes of a prolonged useful life.

PROBLEM OF SURVIVAL IN HYPERTENSION

The ultimate test for any treatment of hypertension is whether or not it alters the progressive and finally fatal course pursued by a great percentage of the cases. Our recent study of patients who had been operated upon five to thirteen years previously showed that 80 per cent of the entire series were still living at the end of five years, 57.3 per cent after ten years and 41.2 per cent after thirteen years. It must be remembered that the great majority of these patients had progressed to very serious constitutional involvement of the heart, kidneys or cerebral vessels before operation. The operative mortality of our entire series is now 1.5 per cent. Comparing the above figures with the few available statistics on the survival of hypertensive patients under the best medical management furnishes strong support for our belief that a bilateral supradiaphragmatic splanchnicectomy offers the very best form of treatment for the patients with progressive hypertension.

MALIGNANT HYPERTENSION

The patient showing definite papilledema, usually with hemorrhages and exudates, and with a high systolic and diastolic pressure, and frequently with evidences of severe cardiac and renal damage presents an absolutely hopeless prognosis from the medical standpoint. In the Keith, Wagener, Barker[7] series of 146 patients with malignant hypertension only 21 per cent were living at the end of the first year. A similar group operated upon with a supradiaphragmatic splanchnicectomy had in contrast a survival of 64 per cent at the end of one year.[17] Those treated medically had a survival of only 12 per cent at the end of the second year while those treated surgically had a survival rate of 50 per cent after the same length of time. After four years only 2 per cent of the medically treated patients were alive contrasting with 33 per cent survival of the surgically treated patients. At the end of five years only one of 146 medically treated patients was alive, while thirty-one (21.6 per cent) of 143 cases were living five years or more after splanchnicectomy. One case with definite preoperative evidence of a severe, malignant hypertension is now living thirteen years postoperatively and the blood pressure has been maintained at a normal level.

PREGNANCY FOLLOWING SPLANCHNICECTOMY

Pregnancy presents a very serious problem in hypertension. Frequently the hypertension is present before the patient becomes pregnant but in some cases it apparently develops as a result of pregnancy. Ordinarily the hypertensive female who becomes pregnant is much less likely to go through child-bearing successfully than is the woman with a normal pressure. She seems markedly vulnerable to the toxemia of pregnancy. Also it has been estimated that about 25 per cent of women

who have a toxemia of pregnancy have a persistent postpartum hypertension.[3] We have operated upon twenty-eight hypertensive females who subsequently experienced thirty-four pregnancies.[19] Eighteen of these women had maintained normal pressures following their splanchnicectomy and before they became pregnant. Seventeen of these gave birth to eighteen living infants and fifteen of the patients were still maintaining normal blood pressure levels at intervals averaging 2.7 years following delivery and 6.3 years following splanchnicectomy. Of the 10 remaining patients whose blood pressure had not been reduced to normal only two were delivered of living infants at full term. None of the women with normal blood pressures following operation and prior to pregnancy developed a toxemia of pregnancy and none suffered any apparent damage to either the heart or the kidneys. Twelve of the women with normal pre-pregnancy blood pressures went through thirteen full term pregnancies and maintained normal blood pressure levels throughout. Mild to moderate elevations were noted during fourteen pregnancies. These included the patients who had an elevation of blood pressure at the onset of pregnancy. In five of these, living infants were delivered at full term. In only four cases (these had elevated pressures at onset of pregnancy) did the blood pressure reach or exceed the preoperative level and in each case the pregnancy was interrupted.

It is evident from the above figures that if a woman with hypertension desires to have a child she should first have a splanchnicectomy. If then her blood pressure comes down to normal and remains so for a year, we think it is safe for her to proceed with child-bearing. Her chances are excellent for delivery of a normal infant and she has little to fear from a possible toxemia of pregnancy or from any late postpartum sequelae associated with her previous hypertension. We have also performed a splanchnicectomy upon five women during the second trimester of their pregnancies because of exceedingly high blood pressures and symptoms of the toxemia of pregnancy.[18] It is generally advised to interrupt such a pregnancy. However, this may not be necessary if a splanchnicectomy is performed. In two of the five cases brilliant results were obtained. In both the blood pressure returned to normal, the toxemia disappeared and normal living infants were delivered at full term. In these two women the normal blood pressure levels have been maintained for four years and two years, respectively, since the delivery. In the other three cases the splanchnicectomy apparently had no influence on the toxemia but in one of these cases the blood pressure levels since delivery have been significantly reduced.

RELIEF OF INCAPACITATION

Incapacitation, either because of the severe symptoms of hypertension or because of organic changes in the heart or the kidneys, has been significantly relieved in a large number of our patients by splanchnicectomy.[14] The ability of these patients to resume gainful work following operation is one of its most gratifying results. In a follow-up study covering seven years, 55.5 per cent of the patients who had been incapacitated had had complete recovery from such incapacitation and had returned to their former occupations. Many others had shown so much improvement that they were again employed although not at their original jobs. This makes a total improvement of 81.3 per cent. As a rule the patients are able to return to work within a few weeks following operation.

CASE REPORTS

The following three case histories are given to illustrate typical results obtained by bilateral supradiaphragmatic splanchnicectomy in patients with malignant hypertension, severe angina and pregnancy following splanchnicectomy.

Case. 1. *Malignant hypertension:* V. E., age twenty-two, was known to have an elevated blood pressure for three years. On entrance she complained of severe headaches, swollen

ankles and blurred vision. The patient had developed severe precordial pain in the left arm, nausea and vomiting, and had been confined to bed for eight months because of the severity of her symptoms. In spite of this long rest her blood pressure averaged 280 systolic, 190 diastolic in each arm. Funduscopic examination showed a severe neuroretinitis with early papilledema, flame-shaped hemorrhages and cotton wool patches. There was localized angiospasm and arteriovenous compression. The electrocardiogram showed inverted T waves in leads II and III. She had a proteinuria of 0.12 per cent and a moderate number of red blood cells and casts in the urine. Water concentration and urea clearance were normal. On June 22, 1934, a bilateral supradiaphragmatic splanchnicectomy was performed. She was discharged on the fourteenth postoperative day.

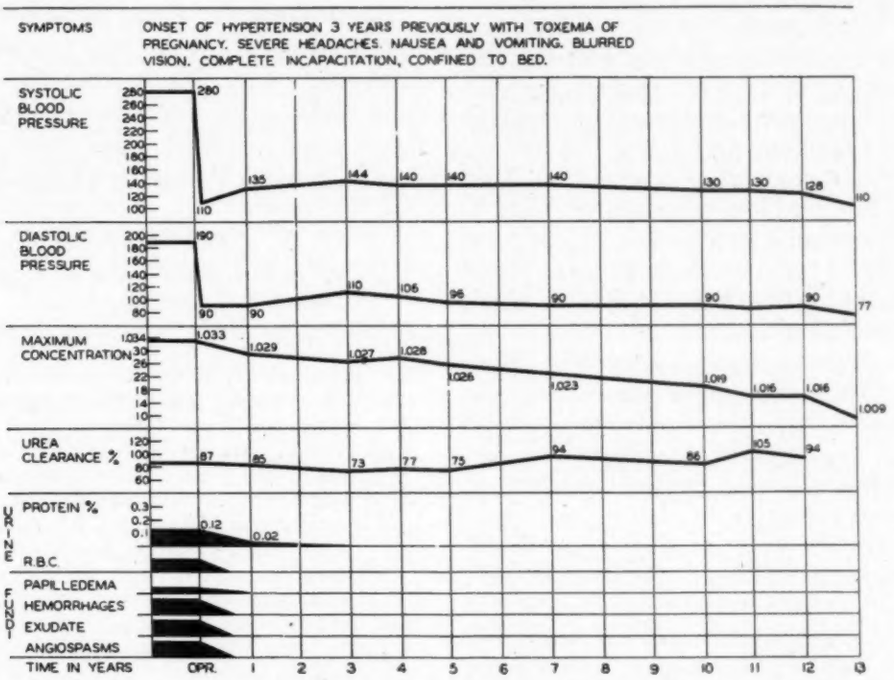

FIG. 1. Chart illustrating results of bilateral supradiaphragmatic splanchnicectomy and lower dorsal sympathetic ganglionectomy during a thirteen-year postoperative period. (Case 1.)

An examination two months later showed that the retinal hemorrhages, exudates and papilledema had completely disappeared. The blood pressure was 145 systolic, 90 diastolic. One year postoperatively her blood pressure was 135 systolic, 90 diastolic. She was completely asymptomatic. Ocular fundi examination was entirely normal. She was examined at yearly intervals with similar findings. At the end of ten years her blood pressure was 130 systolic, 90 diastolic. She was still entirely free of symptoms. Electrocardiogram and heart size were normal. At no time had there been any evidence of retinal disturbance. Twelve years postoperatively her blood pressure was 128 systolic, 90 diastolic. She was still completely asymptomatic. Electrocardiogram and heart size were normal. Thirteen years following operation the blood pressure was still normal at 110 systolic, 77 diastolic. (Fig. 1.) Funduscopic examination, electrocardiogram and orthodiagram were all normal. She was entirely symptom-free.

CASE II. *Hypertension with severe angina pectoris:* M. S., age forty, entered the hospital on September 2, 1941, complaining of episodes of excrutiating pain across the upper chest always related to exertion and disappearing five to fifteen minutes after rest. She had been a known hypertensive for ten years. For the past month she had had from two to six episodes of very severe angina pectoris daily. Her blood pressure averaged 230 systolic, 150 diastolic. Funduscopic examination showed moderate arteriosclerosis and localized angiospasms. There were no hemorrhages or exudates. Electrocardiogram showed deeply inverted T waves in leads I and II and in chest leads evidence of anterior myocardial infarction. Heart size was normal. She

was continued at bed rest and averaged two anginal seizures daily. On September 18, 1941, she had a protracted episode of very severe substernal pain requiring morphine and the following day her electrocardiogram showed prominent T waves in leads II and III and evidences of a new posterior myocardial infarction. The blood pressure remained high. On October 17, 1941, in spite of the fact that she had had two comparatively recent myocardial infarcts a bilateral supradiaphragmatic splanchnicectomy was performed and she had an uneventful postoperative course. Five years postoperatively her blood pressure was 164 systolic, 114 diastolic. Electrocardiogram showed prominent Q waves in leads II and III. Heart size was normal. She has not had a single anginal attack since operation.

Case III. *Pregnancy after a splanchnicectomy:* J. T., age twenty-three, had a known hypertension of two years. Blood pressure averaged 230 systolic, 145 diastolic. Funduscopic examination showed occasional localized angiospasm and marked attenuation of the retinal arterioles. One cotton wool exudate was noted. Electrocardiogram and heart size were normal. The kidney function tests were normal. On April 20, 1938, a bilateral supradiaphragmatic splanchnicectomy was performed. Two years postoperatively her blood pressure was 126 systolic, 82 diastolic. There had been complete relief of the very severe preoperative headaches. Five years after splanchnicectomy she completed a full term pregnancy with a normal living infant. Her blood pressure had remained normal throughout the pregnancy. There had been no albumin in the urine. Seven years after operation her blood pressure was 118 systolic, 86 diastolic. She was completely free of symptoms; the electrocardiogram and heart size were normal. Kidney function was also normal.

SUMMARY

The symptomatology of hypertension is discussed at length and its possible bearing on prognosis given. The etiology of hypertension is considered, especially in reference to its surgical treatment.

A bilateral supradiaphragmatic splanchnicectomy is recommended as the procedure of choice in patients showing a progressive type of hypertension or in those who have already reached a high level.

The results of careful studies over a long period show significant improvement as measured by a reduction in the blood pressure, improvement of ocular, renal and cardiac status, alleviation of symptoms, relief of incapacitation and a probable prolongation of life.

Approximately 2,000 patients have been operated upon by this one procedure at the University Hospital during the past fourteen years and a sufficient number of these have been studied over a long enough period to make definite statements possible.

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TUMORS OF THE SPINAL CORD

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IN recent years, with the discovery that many of the heretofore unexplained pains in the upper and lower extremities were in reality due to protruded intervertebral disks, other intraspinal lesions have been overshadowed and so may be overlooked in the making of a differential diagnosis. The reason for such a statement is that neurologists and neurosurgeons are seeing an increasing number of patients who have been treated for protruded intervertebral disks, only to find that the symptoms have persisted rather than abated. Much to the chagrin of everybody concerned, tumors of the spinal cord subsequently have been removed at operation in such instances, and the symptoms have been relieved thereby.

Intraspinal lesions which produce symptoms of weakness, numbness and pain involving the neck and shoulders, thorax, abdomen, pelvis and extremities create a fascinating and stimulating clinical problem. These lesions may vary from inflammatory adhesions and scars to vascular anomalies and varicosities. They may consist of tumors arising from the bone, fat, cartilage, fibrous tissue and nervous tissue of the extradural space. These tumors may arise also from the arachnoid, blood vessels and nerves of the intradural, extramedullary region, and they may arise from the spinal cord itself, originating either in the fibrous or the neurogenic tissue.

The surgical approach to intraspinal lesions dates back to the time of Hippocrates, but the classical description of the development of symptoms is contained in the report of the first successful removal of a tumor of the spinal cord. Dr. William Gowers and Sir Victor Horsley published this report in the *Medico-Chirurgical Transactions* in 1888, and it is so succinct, so comprehensive and so stimulating that even today it remains a classic.

Since Horsley's epoch-making operation, the diagnosis of tumor of the spinal cord has been made in many cases and the tumors have been successfully removed. Even with the experience gained in hundreds of these cases, however, there still remains an interval between the onset of pain and the development of other symptoms, during which time many diagnoses may be hazarded, and different varieties of treatment may be instituted.

The medical profession at large has learned to accept the fact that when the diagnosis is made early and the tumors are completely removed, excellent clinical results may be obtained. But early diagnosis often is difficult, especially when the only symptom is intermittent pain, sometimes projected to some definite, localized region in the thorax, abdomen or extremities. When the pain is associated with paresis or anesthesia, an organic neurologic lesion is suggested immediately. However, patients sometimes require relief of pain before any associated motor or sensory defect can be determined, and that pain may so simulate the pain of other lesions in the body that radical therapeutic measures are instituted which prove to be ineffectual. Consequently, patients suffering from early pain caused by intraspinal tumors have had gallbladders, appendixes, fallopian tubes and uteri removed, yet, even when a definite pathologic condition could be demonstrated, the pain has recurred. The probability that such operations would be carried out at first seemed rather remote; but in a review of a large series of cases of

proved tumors of the spinal cord, it was found that such measures to relieve the early symptoms of pain actually had been taken in about 10 per cent of the cases, and that the patients concerned ultimately had undergone removal of tumors.

Instances in which such operations were carried out not only represented attempts to relieve pain which, originating from lesions in the spinal cord, was projected to certain other regions, but also demonstrated the possibility of coexisting pathologic change which might be symptomless. Thus, for example, cystic ovaries may be discovered in cases in which pain is projected to the pelvis, and likewise, fibromyomas of the uterus can be demonstrated in cases of painful back associated with pain projected to the pelvis. Evidences of inflammatory processes about the appendix and gallbladder can be demonstrated in cases in which pain is projected to the right lower and right upper abdominal quadrants. But the unsatisfactory attempts to relieve the pain should suggest that the underlying pathologic process involved the spinal cord.

SYMPTOMS

The symptoms of intraspinal lesions have been divided by Oppenheim and by Frazier into three stages, which the authors designated as "cycles." The *first cycle* is the root, or pain cycle, which usually is the longest in the period of the disease. It is not uncommon for patients suffering from root pain to present themselves to their family physician for examination complaining of rheumatism, neuritis and miscellaneous abdominal disturbances. Physicians and surgeons may be tempted to treat these complaints without making a thorough neurologic examination.

Woltman has stressed the point that almost no pain is pathognomonic, but that a given disease may be accompanied by many different kinds of pain; and that although pain may be the only symptom, yet much may be learned by inquiry as to the mode of onset, the duration and the intensity. Pain is a subjective complaint. Pain is something that cannot be seen or palpated, which means that cautious interpretation of what the patient has to tell is necessary. Seven points at least should be determined regarding each complaint of pain: (1) situation, depth and projection, taken together, (2) frequency, (3) duration, (4) character, (5) intensity, (6) progress and (7) associated symptoms.

In the making of a differential diagnosis between pain caused by intraspinal lesions and pain caused by organic lesions of the thorax, abdomen and extremities, many procedures of diagnostic value are available. None of these can begin to compare with a careful and thorough neurologic examination. For such an examination it is necessary to have the patient disrobe completely. The physician must consider any change from normal in the many reflexes, in the response to cutaneous stimulation by touch, heat and cold, and in the strength of the muscles. Roentgenologic examination of the spinal column frequently is of great value in demonstration of the presence or absence of changes in the bony structure caused by inflammation, previous trauma, erosions and tumors. Examination of a specimen of cerebrospinal fluid obtained by lumbar puncture is one of the most valuable diagnostic procedures. The physical, chemical and cytologic characteristics of the fluid may contribute the evidence necessary for diagnosis of an intraspinal lesion. Additional examination of changes in pressure, after an increase in intracranial pressure resulting from compression of both jugular veins, determines the presence of subarachnoid block, which prevents free circulation of the fluid in the subarachnoid space. This sign, coupled with the pathognomonic pain of tumor of the spinal cord, frequently is the means of arrival at an early diagnosis in cases in which an intraspinal lesion is suspected.

In the presence of pain and positive results of examination of the cerebrospinal fluid, it is often very difficult to localize the lesion of the spinal cord sufficiently ac-

curately to enable relief to be given by surgical means. As a means of further differential diagnosis, iodized oil can be used. When this opaque substance is injected within the subarachnoid space, it collects at the level of the block, and this level can be visualized by means of roentgen rays. It is often important to make a very careful neurologic examination after the removal of cerebrospinal fluid, inasmuch as levels at which sensory disturbances appear may become apparent, reflex changes may take place and muscular weakness may occur. With all these diagnostic procedures at the physician's command, it still may be impossible to demonstrate any signs of a neurologic lesion. In such an instance, examination should be repeated at frequent intervals before any drastic therapeutic measures are indicated for the relief of pain in patients suspected of having intraspinal lesions.

We often refer to organic lesions as producing characteristic symptoms which should arouse a suspicion of the underlying lesion. For instance, we say that tumors of the brain produce the three cardinal signs of headache, nausea and vomiting, and increased intracranial pressure. So, also, we speak of intraspinal lesions as giving rise to characteristic symptoms. Pain is the outstanding symptom of intraspinal involvement of nerve roots, and may be characteristic, sometimes being preceded by symptoms of irritation such as paresthesia, hyperesthesia or complete anesthesia in the area supplied by the root or roots. The pain of an intraspinal lesion may precede any other symptom by months or years; it may be constant or intermittent; its chief characteristic is that it occurs when the patient is at rest and is relieved by exercise. The character of the pain is almost pathognomonic since it persists in a localized area and extends over the same nerve roots. It is usually lancinating and is aggravated by coughing, sneezing, lifting and straining at stool. It invariably awakens the patient from four to six hours after retiring. It often becomes so severe as to compel the patient to walk the floor or to sleep in a sitting position. The mechanism of the production of this pain apparently is brought about by the ball-valve action of the tumor, which is forced downward by the increased pressure of the cerebrospinal fluid above the tumor, thus producing traction directly or indirectly upon the nerve roots. Unfortunately, many of the patients are treated for neuritis, muscular rheumatism or syphilis, and some have even been called "hysterical." The importance of the recognition or suspicion of the first or painful phase in the development of spinal cord tumors was emphasized in a recent survey in which 10 per cent of the patients presenting root pain had been operated upon for some thoracic or abdominal lesion other than that of intraspinal tumor.

In the discussion of tumors of the spinal cord it is usual to consider the tumors as they occur anatomically. When the tumor occurs in the *cervical region* of the spinal cord, the pain may be in the neck or it may extend to the shoulders. The pain may be increased by laughing, sneezing and coughing, and may be accompanied by some rigidity of the neck. Tumors in the upper part of the cervical region may produce symptoms similar to those of involvement of the medulla oblongata. Paralysis of the diaphragm may occur when the fourth cervical segment is involved. Tumors of the lower part of the cervical enlargement may cause atrophic paralysis of the small muscles of the hands. The oculopupillary symptoms (contraction of the pupil, narrowing of the palpebral fissure and enophthalmos) are present on the side of the lesion.

Tumors which are compressing the *thoracic segment of the spinal cord* usually cause pain which frequently simulates that of intercostal neuralgia, cholecystitis or cholelithiasis, renal colic or appendicitis. The zone of hyperesthesia is above the level of the lesion.

Tumors situated in the *lumbar and sacral portions of the spinal cord* present

difficulties in differential diagnosis because a neoplasm in this part of the vertebral canal can compress the fibers of the cauda equina and produce practically the same symptoms. Symptoms generally are those produced by compression of the spinal cord. Pain is outstanding, frequently is diagnosed as "sciatica," and may be present for months and years before there are any localizing signs. Functions of the bladder and rectum are interfered with early in tumors of the conus medullaris. These tumors do not cause paralysis unless they grow to such an extent as to compress the roots of the cauda equina. Disease of the conus medullaris is characterized by rapid development, absence of pain, appearance of anesthesia of an associated character, motor irritative phenomena such as fibrillary twitchings, and general and rapid development of paralysis of the bladder, rectum and intestinal musculature.

For convenience in discussing the symptomatologic aspects of intraspinal lesions, a *second cycle* or phase has been described. The symptoms which develop in this second phase differ from those of the first in that neurologic evidence of compression of the spinal cord now becomes evident. These symptoms may develop simultaneously with the existence of pain or they may develop without the presence of pain. If the tumor is situated anterolaterally, the symptoms will progress and produce the classic Brown-Séquard syndrome. This is the characteristic symptomatology of the second phase. It consists of homolateral paralysis or paresis of the muscles below the level of the lesion, with a loss of, or diminution in, sensation of pain and temperature on the opposite side. This syndrome is common in cases of extramedullary tumors because most of these tumors exert their pressure on the anterolateral and posterolateral aspects of the cord. If the posterior columns of the spinal cord are interfered with, deep sensibility is decreased, and consequently ataxia in the extremities and below the lesion occurs. Sensory disturbances caused by compression of the spinal cord develop in an ascending manner; after removal of the lesion normal perceptive powers are restored in a descending sequence.

Tumors of the upper and lower ends of the spinal cord may present certain peculiar features. The crossing of the pyramidal tracts at the anterior aspect of the cord in an exposed situation may result in quadriplegia unaccompanied by any sensory disturbance. The sensory disturbance associated with high cervical tumors may be limited to the lower extremities; it may fluctuate from time to time and from place to place, probably because the relatively large cervical canal permits shifting of the pressure. The proximity of the foramen occipitale magnum may bring about signs of a tumor of the brain, such as a projectile type of vomiting and choked optic disks.

When a tumor occurs at the lower end of the spinal cord, other difficulties may be encountered. The relative shortening of the cord incident to growth and the emergence of the roots through the anterior foramina of the sacrum often make it extremely difficult to determine whether there is a tumor of the cord or the cauda equina, or a tumor situated in the pelvis. The objective findings may be the same.

Again, the *third cycle* or phase in the symptomatologic aspects of intraspinal tumors may be characteristic of the progress of the disease. Paralysis occurring below the level of the tumor comprises this third phase. It is caused by extreme compression of the cord. The paralysis usually is complete, for both motor and sensory functions and trophic disturbances are present, as well as definite loss of both vesical and rectal sphincter control.

EXAMINATION

After the taking and recording of a history, a detailed general, as well as a neurologic, examination is necessary. This should include such special examinations as spinal puncture and the making of roent-

genograms of the spinal column, with or without the introduction of iodized oil.

General Examination. In the presence of all suspected intraspinal lesions, close inspection of the skin of the body is necessary for evidences of cutaneous fibromas or the pigmented patches of multiple neurofibromas (von Recklinghausen's disease). Primary malignant lesions of the abdominal and pelvic viscera should be kept in mind when patients are elderly, and in such instances the urine should be examined for evidence of renal tumors. Dumb-bell tumors have been found with roentgenologic evidence of tumors within the thorax indicative of this type of compression of the spinal cord.

Roentgenologic Examination. Roentgenograms should be made of the anterior, posterior and lateral aspects of the vertebral column. These roentgenograms should be supplemented by stereoscopic and oblique views made at the level suspected clinically. According to Camp and Adson evidence of erosion of the vertebral pedicles, laminas and lateral and spinous processes resulting from pressure usually is discernible before comparable evidence is apparent in the bodies of the vertebrae. In general, the roentgenologic evidence of changes caused by tumors of the spinal cord consists of erosion secondary to direct pressure, invasion by the tumor, destruction brought about by benign or malignant tumor of the bone, metastatic disease and hyperostosis.

Camp, in a review of cases, reported that osseous changes which directly localized the lesion were observed roentgenographically in 30.3 per cent of the cases. He divided tumors of the spinal cord into three groups roentgenologically.

The first, and fortunately the most predominant group, are benign tumors arising from the soft tissues within the spinal cord. The most common in this group include neurofibroma, endothelioma, hemangioma, angioma, lipoma and dermoid cyst.

The second group of tumors classified roentgenologically consist of malignant tumors arising from the soft tissues within the spinal canal. These usually are metastatic and as a rule are diagnosed by means of roentgenograms.

The third group comprise the benign tumors of the vertebrae, including osteomas, osteochondromas, chondromas, fibrochondromas, giant-cell tumors and hemangiomas.

Roentgenologic diagnosis can be made in the presence of primary infections of the vertebra, such as tuberculosis, osteomyelitis, actinomycosis and coccidioidal granuloma, which produce destructive areas in the vertebra, together with involvement of intervertebral disks.

Of distinct value is roentgenologic examination of the intervertebral canal with contrast media. In addition to the roentgenologic evidence of tumors, which is apparent in routine examination of the cord, roentgenoscopic and roentgenographic study by the use of radiopaque oil furnishes considerable information. Injection of 5 cc, of iodized oil into the subarachnoid space. either through cisternal puncture or by means of lumbar puncture, allows visualization of the patency of the subarachnoid space under the roentgenoscope. Roentgenoscopic examination of slowly moving oil is superior to study of the roentgenogram, since the physician often is able to see the diversion of the oil current around the tumor, although roentgenograms should be made for confirmation of the level of the lesion. Intramedullary tumors are identified by a division of the oil into two currents. After the oil has been injected, the patient should be placed prone on the roentgenoscopic table and the flow of oil should be observed with the patient in the various positions, from the horizontal to the perpendicular. Experience with the use of radiopaque oil in the diagnosis of tumor of the spinal cord has indicated that it should be used only in conjunction with a complete examination, and that the oil should be removed immediately after the need for it has passed. The iodized oils most frequently used are panto-

paque and lipiodol. Pantopaque is employed more often in the differential diagnosis when protruded intervertebral disks are suspected. Camp and his colleagues have called attention to the fact that when a tumor is suspected to be situated above the conus medullaris, the use of lipiodol allows for a more nearly exact examination.

Neurologic Examination. When intraspinal tumor is suspected, there is no examination so important as a complete neurologic examination. The information elicited by a detailed testing of reflexes, muscle strength and tonus, sensory acuity, as well as gait, co-ordination and balance, tends to distinguish between degenerative diseases and the compression syndrome of intraspinal tumor.

Since the spinal nerves carry both sensory and motor impulses, a lesion of these will manifest itself by disturbance of all forms of sensation and motility. Involvement of a posterior root alone will result in disturbance of all forms of sensation; involvement of an anterior root alone will produce only a motor disturbance. After entry of the posterior root into the cord, however, the various forms of sensation pursue different paths, so that a lesion situated within the spinal cord might not affect all forms of sensation.

Fibers which carry sensations of pain and temperature are closely associated. They may be regarded as taking the same course. They cross almost immediately to the opposite side of the cord in the anterior white commissure, and ascend to the brain in the lateral spinothalamic tract.

Tactile stimuli take two pathways: one proceeds up the posterior column on the same side of the cord, but crosses to the opposite side after it reaches the brain stem; the other crosses almost immediately and ascends in the anterior spinothalamic tract of the opposite side. Therefore, a lesion of either half of the cord alone will not abolish tactile sensation completely.

Motor impulses arise in the brain, leave the pyramidal cells of the prerolandic area, and pass downward in the pyramidal tract, which crosses to the opposite side about 1 cm. below the pons cerebelli and descends in the lateral column of the cord. A lesion of the pyramidal tract results in the form of paralysis seen in ordinary hemiplegia. The muscles are not wasted; they are hypertonic, and the tendon reflexes are more active than is normal. The plantar response elicited by the Babinski method usually is extensor; that is, when the sole of the foot is stroked, the great toe turns up.

The motor impulses leave the pyramidal tract and are relayed to the cells of the anterior horn, or lower motor neuron, on the same side. A lesion of the lower motor neuron results in a form of paralysis differing in many respects from that caused by involvement of the upper motor neuron and resembling that seen in poliomyelitis. The muscle fibers supplied by the corresponding neurons are weak and atrophied, and the tendon reflexes are reduced to a point commensurate with the amount of muscle substance remaining.

Since, in compression of the spinal cord, one side usually is affected a little before the other, the so-called Brown-Séquard syndrome is produced. The cardinal features of this syndrome are: (1) homolateral paralysis below the given level, caused by involvement of the pyramidal tract, (2) impairment of pain and sensation of temperature of the opposite side below the level in question and (3) preservation of tactile sensibility on both sides.

The segmental distribution of cutaneous sensation is very orderly. The various fields of sensation supplied by the different segments of the spinal cord can be mapped out and shown to be running in long bands down the arms, in circles about the trunk, in bands down the legs, and ending in the concentric rings around the anus. Actually, these segments overlap each other like shingles on a house, so that two or three adjacent roots would have to be destroyed before the area of anesthesia could be demonstrated.

Much interest has been evidenced recently in the determination of the upper levels of lesions and tumors of the spinal cord by means of vasomotor lines of demarcation. Vasomotor lines of demarcation frequently will indicate the level of the lesion before the development of sensory or motor symptoms of focal value. In many instances pilocarpine has been used to stimulate sweating; and the reaction indicates that in cases of intramedullary disease, the sweating response rarely indicates a level of any diagnostic value. In cases of extramedullary disease (tumor), the sweating reaction tends to divide the body into contrasting areas, meaning that there is very little sweating below the level of the lesion. As a rule, in extramedullary disease the segmental level below which sweating is suppressed corresponds with the level below which sensation is diminished. The segmental representation of the reflexes also aids in determination of the site of the involved portion of the spinal cord.

Spinal Puncture. Examination of the cerebrospinal fluid discloses information concerning the physical and hydrodynamic properties and the chemical reactions of the cerebrospinal fluid. Spinal puncture generally is performed at the fourth lumbar interspace. Before any fluid is removed, the intraspinal pressure is estimated by means of a manometer which normally registers between 12 and 15 cm. of water. As soon as the pressure has been estimated, Queckenstedt's test is made. This consists of reading and study of the rate of increase of the cerebrospinal fluid in the manometer after compression of both internal jugular veins. The sudden increase and rapid decrease of the fluid on compression of both internal jugular veins indicates a free flow of cerebrospinal fluid within the subarachnoid space, and rules out the possibility of a compressive mechanism. A slow increase and decrease of fluid or failure of the fluid to rise at all on compression on the jugular veins suggests partial or complete intraspinal block.

Inability to obtain fluid at the fourth lumbar interspace may signify that the subarachnoid space has not been entered, or absence of fluid or the presence of a tumor at this level. In such a circumstance other levels should be used, and it may be necessary to make multiple punctures which may localize the tumor definitely.

Examination of the cerebrospinal fluid with regard to the number and kind of cells and the amount of protein, and also other chemical tests, as well as the colloidal gold curve, may be methods of distinguishing between intraspinal lesions and degenerative lesions, such as multiple sclercsis.

It must be remembered that the presence of partial or total subarachnoid block is not pathognomonic of intraspinal tumor since previous attacks of meningitis, acute myelitis, injuries to the vertebrae or spinal deformities are all capable of interfering with the free flow of cerebrospinal fluid. But it is apparent that subarachnoid block is extremely valuable diagnostically when it is found in conjunction with a history of root pain without inflammation or trauma to the spinal cord.

Differential Diagnosis. The physician must always keep in mind, when a tumor of the spinal cord is suspected, the possibility of intraspinal metastatic lesions. For that reason, a complete general examination is important. Carcinomas of the breast and prostate gland head the list of metastatic lesions to the spinal cord. It must not be forgotten that a primary carcinoma of the breast may have been removed many years before metastasis to the cord gives evidence of its presence. Since carcinoma of the prostate gland may not produce local symptoms, rectal examination should be made.

Meningomyelitis is another condition which may remain localized, and in which there is often a history of antecedent trauma or infection.

The spinal column itself may be the seat of the original disturbance which causes pressure on the spinal cord. This may occur as the result of *Pott's disease, chronic hyper-*

trophic osteo-arthritis, spondylolisthesis, or a slipping forward of a vertebra—usually the fifth lumbar vertebra—on the sacrum, and *Paget's disease.*

Spina bifida may be associated with a superimposed tumor.

In cases of *subacute combined degeneration* of the spinal cord, the lesion, although progressive, as a rule is painless and diffuse.

The diagnosis of *tabes dorsalis* sometimes is made when there are tumors of the spinal cord or cauda equina, probably because the patellar reflexes may be absent, but a complete examination usually indicates the true nature of the disease.

It may be difficult to distinguish *syringomyelia* from an intramedullary tumor of the spinal cord. Aids to correct diagnosis are the characteristic waistcoat type of sensory disturbance, which seems to affect chiefly pain and temperature fibers, since these lie near the central canal; the local atrophy caused by extension of the process here and there into the anterior horns, and the tell-tale scoliosis.

Although *multiple sclerosis* may produce a transverse lesion of the spinal cord, the youth of the patient, the absence of pain and the presence of cerebral signs, such as tremor, scanning speech, nystagmus, optic atrophy, ocular palsy and the characteristic emotional lability generally put the physician on guard.

SURGICAL TREATMENT

Anesthesia. The selection of the proper anesthetic agent to use during operations on the spinal cord depends on the patient and the facilities for administration. Most patients prefer general anesthesia. Ether dropped onto an open mask or over a Magill intratracheal tube is to be preferred to ethylene or nitrous oxide. Paravertebral regional anesthesia produced with procaine hydrochloride and epinephrine minimizes the amount of bleeding, but cannot be used for hypersensitive patients. Tribromethyl alcohol (avertin) has been used successfully, although occasionally an idiosyncrasy toward the drug has proved alarming.

Operation. Once the diagnosis of compression of the spinal cord by a tumor of the cord has been established, the treatment is essentially surgical, because the object is relief of compression of the spinal cord. The surgical mortality rate associated with removal of tumors of the spinal cord is less than 4 per cent. Exposure of the spinal cord at operation is accompanied by so little risk that it can be carried out routinely in the presence of dysfunction of the spinal cord associated with a distinct sensory level and subarachnoid block, or when the level has been established by means of iodized oil. Good exposure of the cord is an essential factor, and, of course, the size and extent of the tumor necessarily control the extent of the laminectomy. The mechanical removal of the spinous processes and laminas is not so important as is close observation of the tissue removed. The texture of the removed bone should be scrutinized carefully for evidences of any changes, either of absorption or of overgrowth. Then the epidural space requires critical survey for signs of neoplastic or inflammatory change. If the results of exploration have been negative up to this point, the dura should be carefully examined for any abnormalities of pulsation. Absence of pulsation means that one of two conditions is present: either the compression is situated above the laminectomy opening or the exposed dura lies just over the tumor. Gentle palpation of the dura rules out the latter condition; and if there are no signs of compression, the laminectomy must be carried cephalad until there is a definite pulsation of the dura. During the operation, it must be kept in mind that accurate as the localization has been, there is always danger that the level of the abnormality, as determined before operation, may be one segment lower than the actual compression of the cord.

Extramedullary tumors are apparent by this time in the carrying out of the operation, and should no evidence of compression be found to be caused by extradural

lesions, the dura can be opened and the cord examined. A silver probe or soft rubber catheter can be gently inserted intradurally to eliminate the possibility of an obstruction, either above or below.

Intramedullary lesions, or those situated within the spinal cord, may cause symptoms of compression. The majority of these lesions involve a discouraging prognosis, yet cysts have been evacuated, benign tumors have been completely removed and recovery has resulted from the decompression incidental to the laminectomy.

There are certain non-neoplastic lesions which have to be kept in mind in the performance of laminectomy for compression. Pachymeningitis brought about by tuberculosis, syphilis or other chronic inflammatory lesions may prove to be the cause of the compression. Chronic cystic arachnoiditis may be encountered when the dura is opened. Varicosities of the meningeal vessels may simulate tumors, although such a condition may be found adjacent to tumors, making the diagnosis of varicosity possible only after the presence of a tumor definitely has been excluded. The surgical and pathologic aspects of compression of the spinal cord are so closely interwoven that until a complete examination of the vertebrae, meninges and cord has been carried out, the operation should not be terminated or the diagnosis completed.

A linear incision is made in the skin immediately over the spinous processes which are to be removed. The incision should extend at least one segment or two segments above and below. Subperiosteal dissection of the periosteum of the spinous processes and the laminas in conjunction with the erector spinae muscles controls the bleeding and permits more efficacious closure than would incision of the muscle attachments. After the muscles have been dissected from the laminas and are held in place by self-retaining retractors, the bone is carefully removed, resection being carried out fairly wide on each side to prevent subsequent compression on the cord by calluses which may develop. Prominent spinous processes above and below the site of laminectomy should be resected in order to avoid any undue prominences in the wound. In the removal of laminas over a tumor, extreme care must be used to avoid additional pressure on the cord and accidental trauma or interference with the circulation by sponging and dissection. If the tumor is situated anterolaterally or anteriorly, it is better to section it and remove it piecemeal rather than to attempt to force it out intact at the risk of injury to the cord. If, after careful inspection of the meninges, there is no evidence of an extramedullary tumor and the enlargement of the cord indicates a tumor arising within the cord, a longitudinal incision to one side of the midline can be made; if the tumor is cystic, the contents of the cavity can be aspirated and sometimes the wall of the cyst can be removed. Solid tumors sometimes can be totally or partially removed, but additional trauma is to be guarded against. In closure of the wound, the dura should be sutured, unless by so doing pressure would be exerted on the cord. The bases of tumors attached to the dura should, of course, be resected. This will leave a defect in the dura. When it is impossible to close the dura, animal membrane or polythene should be used to cover the defect, so that the entrance of blood into the subarachnoid or dural spaces can be prevented. The muscles should be approximated by interrupted sutures of catgut, as should also the fascial planes and skin. Drainage of laminectomy wounds should be done at the discretion of the surgeon; and although it has been considered dangerous from the standpoint of secondary infection, the use of Penrose drainage for twenty-four hours sometimes prevents postoperative hemorrhage which would necessitate a secondary opening.

The fundamental principles underlying laminectomy are the same whether the operation is performed in the cervical, thoracic or lumbar regions. However, wherever possible, unilateral laminectomy should be done in the cervical region to

prevent the slipping of one vertebra upon another as a postoperative complication.

POSTOPERATIVE CARE

After the operation the patient is placed in bed in the horizontal position. Pillows are used to avoid undue pressure on the shoulders and hips. The patient should be turned from side to side and on the abdomen and not on the back, for the latter action would produce pressure on the wound. One of the frequent postoperative complications is inability to void. If, after twenty-four hours, the patient cannot void, one ampule (1 cc.) of prostigmine methylsulfate (1:2,000 solution) should be administered hypodermically, and if this does not cause voiding, the injection may be repeated in four hours. If this fails, one ampule (1 cc.) of doryl (acetylcholine chloride) may be given a trial. However, patients frequently complain of severe abdominal cramps after the use of this agent. If, in spite of the aforementioned measures, there is still no tendency toward voluntary voiding, the patient should be catheterized. If, then, the bladder still does not empty after catheterization has been carried out once or twice, the use of an indwelling catheter for a week or ten days is safer than daily catheterization. This indwelling catheter should be replaced every four or five days, and the bladder should be irrigated twice daily with an antiseptic solution, usually boric acid solution. Each time the indwelling catheter is replaced, the introduction of a few cubic centimeters of a 2 per cent solution of merbromin (mercurochrome) into the bladder before removal of the catheter sometimes will cause a return of vesical function if the patient is allowed to return the drug before another catheter is inserted. Doses of mineral oil or milk of magnesia are administered daily to prevent distention of the bowel or fecal impaction. In addition to this, a daily enema is sometimes necessary. In order to assure satisfactory healing, the patient should be kept in bed for ten days to two weeks. He should be permitted to sit upright in bed for a day or two before he is allowed to be up in a wheelchair or attempting to walk.

The recovery of motor, sensory, vesical, rectal and sexual functions takes place in the reverse order of their development. The time for recovery of loss of motor and sensory functions depends on the character of the tumor and the degree and duration of paralysis. Hard, rounded tumors cause greater injury to the spinal cord and more paralysis than do the soft, elongated tumors. A 25 per cent loss of function usually is recovered within three months; a 50 per cent loss requires from six to twelve months; a 75 per cent loss requires up to eighteen months; and a total loss requires up to two years unless the injury to the cord has been so extensive that recovery will never take place. The removal of intramedullary, infiltrating tumors often results in temporary improvement which may continue for six to seven years.

PATHOLOGIC ASPECTS OF TUMORS OF THE SPINAL CORD

We have already referred to the classical descriptions of the first instance of removal of a tumor of the spinal cord. The lesion in question proved to be an extradural fibroma; consequently the return of the patient's condition to normal was rather speedy. The anatomic situation of the tumor in respect to the meninges is important in the making of the prognosis. However, pathologically, it is important to note the anatomic situation of the tumor with regard to segments of the spinal cord. It is interesting to observe that, anatomically, the majority of tumors of the spinal cord occur in the thoracic region. This might be explained by the fact that the thoracic segments comprise more than half of the entire length of the cord.

Five hundred fifty-seven verified intraspinal neoplasms were classified pathologically and grouped according to situation by Rasmussen, Kernohan and Adson. Neurofibromas constituted the largest single group. Meningiomas, or arachnoid fibro-

blastomas, comprised the second largest group, and the primary distribution of these was found to be in the thoracic region. Ependymomas were noted to be fairly evenly distributed throughout the spinal cord. Half of them, it appeared, arise from the spinal cord proper, and the remaining half arise from the filum terminale. Vascular tumors form a group which includes the hemangio-endotheliomas and hemangiomas. Chordomas were found to occur most often in the sacral region. Sarcomas, under the heading "sarcomas," were included by Rasmussen, Kernohan and Adson in a miscellaneous group of fifty-five sarcomatous lesions consisting of lymphosarcomas, myelosarcomas, giant-cell sarcomas, Hodgkin's disease and osteogenic sarcoma.

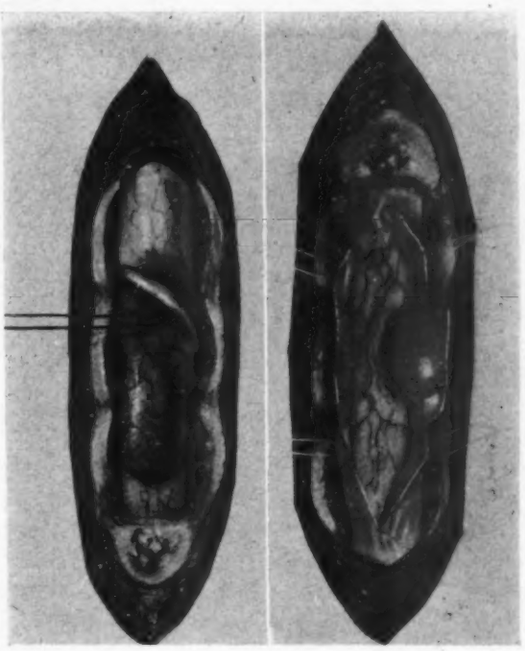

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Fig. 1. Extradural tumor. Such a tumor develops from tissues present in the extradural space; retouched photograph.

Fig. 2. Intradural extramedullary meningioma or endothelioma; retouched photograph.

EXTRADURAL LESIONS

The extradural lesions (Fig. 1) include inflammatory lesions, tuberculosis and tuberculomas, hypertrophic osteitis and metastatic tumors such as carcinomas, fibromyxosarcomas, fibrosarcomas and hypernephromas.

Extradural gliomas are very rare; they probably are extensions, into the epidural space, of tumors of the spinal cord.

Hemangiomas and hemangio-endotheliomas may occur extradurally; they arise from the vessels which normally are found lying extradurally.

Extradural lipomas have been found, and although they are rare except when they are associated with spina bifida, they generally are found in the lumbar region.

Myeloma is, or multiple myelomas are, the rarest of the four types of primary malignant tumors of bone. The lesions constitute a disease of the bone marrow.

Neurofibromas occur extradurally. They belong to the same group as does the perineural fibroblastoma of Penfield. These tumors may occur extradurally or intradurally; they have even occurred in the intramedullary situation. Many neurofibromas within the spinal canal have a tendency to degenerate or become cystic; in a number of instances in the lumbar region neurofibromas have been known to grow to considerable size, eroding the laminas, pedicles and bodies of the vertebrae without producing signs of complete paraplegia. Neurofibromas also produce extradural, intradural and extravertebral tumors, the so-called hourglass or dumbbell perineural fibroblastomas. These may be discovered as tumors within the wall of the thorax, and only on careful examination can any signs of pressure of the spinal cord be elicited. These tumors should be removed very carefully; the intrathoracic mass ought to be removed in conjunction with the intraspinal mass to guard against intraspinal hemorrhages which have occurred to complicate such operations.

INTRADURAL EXTRAMEDULLARY LESIONS

It is interesting to observe that the intradural extramedullary types of tumors comprise approximately half of the entire group of tumors of the spinal cord. Fortunately, it is possible to remove most of

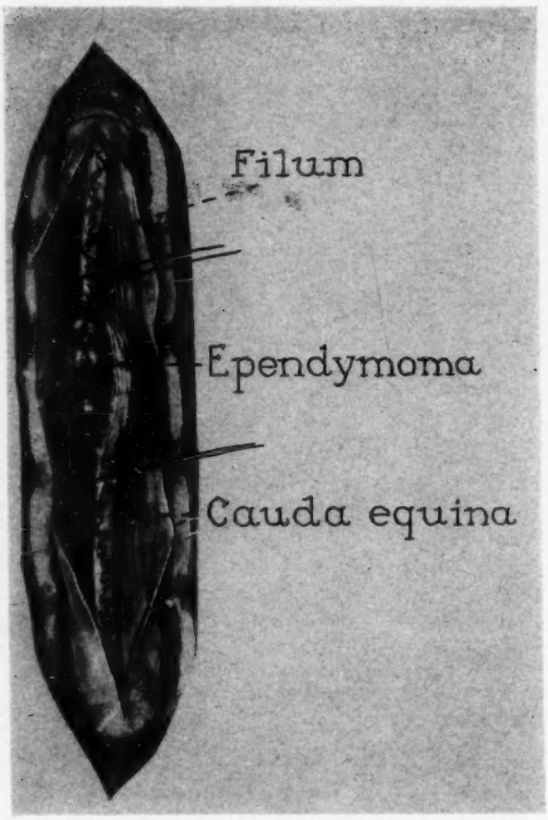

FIG. 3. Ependymoma occurring on the filum terminale producing compression on the cauda equina; retouched photograph.

them at operation, and most of them are benign and do not have a tendency to recur.

The so-called endotheliomas (Fig. 2) are found predominantly in intradural but extramedullary situations. They form the largest single class of tumors of the spinal cord. Fortunately, in view of their numerical incidence, they are associated with an excellent prognosis postoperatively. Although they are benign and encapsulated, these tumors have a tendency to recur if their dural attachment is not removed at the time of operation.

Ependymomas (Fig. 3) are tumors of the spinal cord found attached to the fibers of the cauda equina. They have proved to be tremendously difficult to classify pathologically.

Fibromas and fibrosarcomas may occur within the dura and involve the spinal cord only by compression. They can be completely removed at operation.

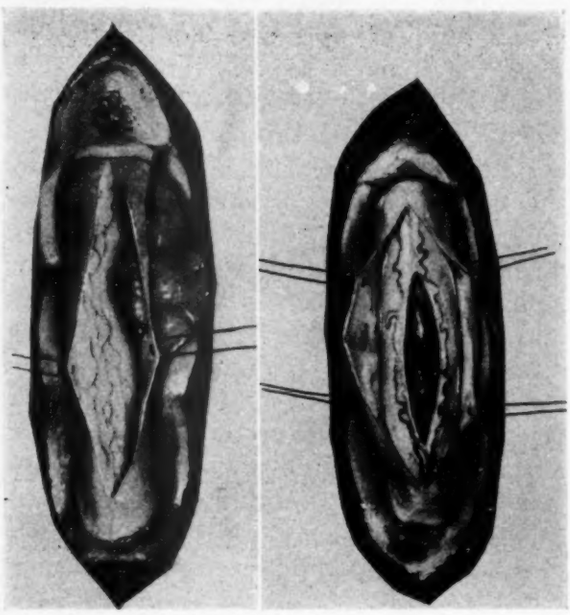

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FIG. 4. Neurofibroma, which may occur either intradurally or extradurally or may be combined as illustrated, showing method of compression on the cauda equina; retouched photograph.

FIG. 5. Intramedullary tumor, showing method of exposure and removal; retouched photograph.

As a cause of compression of the spinal cord, intradural neurofibromas (Fig. 4) were found to be second, in point of number, only to the meningiomas or meningeal fibroblastomas.

INTRAMEDULLARY LESIONS

Thus far consideration has been given herein to extramedullary tumors which produce symptoms by the exertion of compression on the spinal cord. A third group consists of those lesions which involve the spinal cord by growing within its substance. (Fig. 5.) Primary tumors of the cord predominate, but occasionally a benign encapsulated tumor is found which proves to be a neurofibroma, an endothelioma, a lipoma or a fibroma. Moreover, there is always the possibility that there is a metastatic lesion within the cord which proves to be either carcinoma or sarcoma.

It has been found that of the primary tumors of the spinal cord and filum terminale, by far the most common are the ependymomas. However, the types of tumor which are found in the brain also

may be found in the spinal cord. Kernohan has found astrocytomas, astroblastomas, spongioblastomas, oligodendrogliomas and oligodendroblastomas, ganglioneuromas or neuroblastomas in the spinal cord.

SUMMARY AND COMMENT

Intraspinal tumors as a cause of pain and disability have been overlooked in recent years because of the discovery that many of the heretofore unexplained pains in the back and upper and lower extremities actually are the results of protruded intervertebral disks.

Clinically, the majority of intraspinal tumors are benign and can be removed. The possibility of metastatic lesions must be kept in mind, but the age of the patient and a careful general examination will rule this out. Degenerative lesions of the spinal cord also must be ruled out before a definite diagnosis can be made. Careful neurologic examination, including attention to both sensory and motor areas, should be carried out in all cases. Roentgenologic examination of the vertebral column often is of value in differential diagnosis. The introduction of iodized oil within the subarachnoid space has been of immense value in the localization of intraspinal tumors before they have progressed to the point of compression at which definite sensory levels are produced.

Intraspinal operations can be carried out with a minimum of danger. The majority of intraspinal tumors are benign and removable.

Anatomically, intraspinal tumors can be classed as extradural, intradural and intramedullary. Fortunately, the majority of tumors occur outside of the spinal cord, and the symptoms are produced by pressure. Surgical removal of this compression of the spinal cord results in relief of the clinical symptoms. The cardinal symptom of intraspinal tumors is pain, pain which appears intermittently and is increased by coughing and sneezing, pain which occurs after the patient is lying in the prone position, pain which usually causes the patient to arise early in the morning and sit in a chair, and which as a rule is confined to certain dermatomes, indicating the compression of one or more spinal nerves. Compression of the spinal cord produces symptoms, both sensory and motor; generally the so-called Brown-Séquard syndrome is present, with motor symptoms on the ipsilateral side and sensory symptoms on the contralateral side.

It is sometimes difficult to distinguish intramedullary tumors of the spinal cord from tumors of the extramedullary type, although early involvement of the vesical and rectal sphincters as a rule is indicative of an intramedullary type of tumor.

On the whole, it can be said that intraspinal tumors are removable, benign and do not tend to recur. If such a tumor is removed before irreparable changes have taken place in the spinal cord as a result of compression, complete restoration of motor and sensory loss below the level of the lesion should take place.

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TRAUMATIC INTRACRANIAL HEMORRHAGE*

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INTRACRANIAL hemorrhagic collections resulting from injury have been classified upon the basis of an anatomic location within the cranial cavity. The main groups include: (1) Epidural hemorrhage, produced by tearing of the middle meningeal vessels or dural sinuses, (2) subdural collections resulting mainly from disruption of cortical pial vessels, (3) subarachnoid hemorrhage produced by cerebral contusion and laceration and (4) intraparenchymatous hemorrhage due to intracerebral bleeding.

The type of hemorrhage may be correlated to some degree with the nature of the injuring force. When a direct blow or force strikes the non-moving or slower moving head, localized vascular injury is produced, the middle meningeal vessels may be torn, a venous sinus disrupted or a pial vessel ruptured as cerebral tissue is bruised and lacerated. The forces of indirect injury, when the head is decelerated as it rapidly moves against a slower moving or non-moving object, produce more complex, diffuse and combinations of vascular damage. The greater the velocity of the energy involved, the more extensive the pattern of vascular disruption.

The frequency of combinations of both vascular and parenchymatous damage must be emphasized since both the diagnosis and management are influenced by this circumstance. Thus, an extradural hemorrhage may co-exist with cerebral contusions, intracerebral petechial hemorrhages, subarachnoid and subdural bleeding. (Fig. 1.) However, one lesion is usually predominant and its clinical-surgical characteristics are sufficiently typical to warrant separate classification and discussion.

The following classification of vascular lesions is used in this analysis with a review of the pertinent findings in surgically treated and autopsied cases.

1. Epidural hemorrhage
2. Subdural hemorrhage
   (a) Acute type
   (b) Subacute chronic type
   (c) Acute and chronic types in infants
3. Subarachnoid hemorrhage
4. Intraparenchymatous hemorrhage
   (a) Petechial
   (b) Massive
5. Subdural accumulation of spinal fluids

EPIDURAL HEMORRHAGE

The most common type of extradural hemorrhage is of middle meningeal vessel origin. (Table 1.) Epidural hemorrhage from the sagittal sinus is occasionally seen. The latter may occur from depressed, comminuted fractures near the midline of the vault. In one of the cases studied an ice pick perforated the sinus, causing extensive extradural and intradural hemorrhage. A large clot collected between the two hemispheres in this instance and overlay the corpus collosum. Extradural hemorrhage of occipital emissary vein origin was noted in several cases of depressed fracture posterior and superior to the mastoid region. Occasionally the lateral sinus was involved, an occurrence associated with penetrating wounds.

Since extradural hemorrhage is usually of middle meningeal origin, the terms middle meningeal, epidural and extradural hemorrhage are used interchangeably. This type of collection is usually unilateral. Two cases of bilateral extradural clots have been

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seen in this series. One patient was operated upon and the other was seen at the postmortem table.

Etiology. Falls, bicycle accidents and direct head blows are frequently the cause of extradural hemorrhage. In a series of 158

TABLE 1
THIRTY-FIVE OPERATED CASES OF MIDDLE MENINGEAL HEMORRHAGE

State of consciousness	
Lucid interval	13
Unconscious throughout	13
Drowsy and disoriented	8
Short unconsciousness followed by normal conscious state	1
Pupils	
Larger on same side	25
Larger on opposite side	1
Equal	9
Extraocular palsy	
Third	4
Fourth	1
Sixth	1
Focal signs	
Present	30
Not present	5
Vital functions	
Pulse	45–60
Respirations	18–26
Temperature	100–102
Spinal-fluid findings	
Pressure	45–650
Bloody	In 17 cases
Clear	In 1 case
Associated massive lesions	
Subdural hemorrhage	7
Temporo-sphenoidal-lobe clot	4
Subdural accumulation of spinal fluid	2
Recovered	26
Died	9

autopsied cases of cranial injuries, seven out of eleven fatal cases of epidural hemorrhage were the result of such low velocity, direct injuries. High velocity automobile injuries involving indirect forces appear to cause a smaller number of middle meningeal hemorrhages.

Clinical Observations. Although the classic "lucid interval" characteristic of the clinical syndrome of extradural hemorrhage may occur, it is the exception rather than the rule. An already unconscious state gradually deepening is commonly observed. A "lucid interval" may be absent due to co-existing pathologic damage within the cranial cavity. Occasionally hemorrhage from the meningeal vessels superimposed upon a concussive state may be so rapid as to preclude a conscious period.

A frequent clinical finding is dilatation of the pupil on the side of the lesion. Extraocular palsies occur. The enlarging clot may compress the ocular nerve or nerves as they traverse the superior orbital fissure or the pressure may occur by the bulging inner aspect of the temporal lobe medially, involving and interrupting the nerve or nerves in their intracranial course from brain stem to the cavernous sinus. A dilated pupil without other signs of oculomotor paralysis may also be attributed to the above described etiology or to paralysis of the cortical pupillary constricting mechanism. In favor of a peripheral mechanism is the fact that when a clot is located at the base, pupillary manifestations are the rule. (Fig. 2.) When the clot covers the parietal and temporal areas, the pupils are likely to be equal in size. (Fig. 3.) A dilated pupil is usually on the side of the lesion. A constricted pupil on the side of the lesion was seen only once in this series. Paralysis of the third nerve was observed in four cases.

Increasing weakness and paralysis result from compression of the clot over the motor centers. The paralysis is usually on the opposite side from the clot. Occasionally it may be seen on the same side as the clot. In the untreated case the patient fails rapidly; the deep tendon reflexes become diminished, then lost; the plantar reflexes become unobtainable; the corneal response becomes imperceptible; generalized or Jacksonian convulsions may occur. The importance of constant observation cannot be overemphasized since these changes may follow one another in rapid succession with eventual fatal outcome.

Roentgen Observations. Survey roentgen studies of the skull may be of assistance in the diagnosis of an extradural hematoma. A fracture line crossing the temporoparietal region suggests this possibility. (Fig. 4.) Depressed temporal fractures may be associated with hemorrhage; five cases occurred in this series. Because of this association, patients with simple depressions, even if

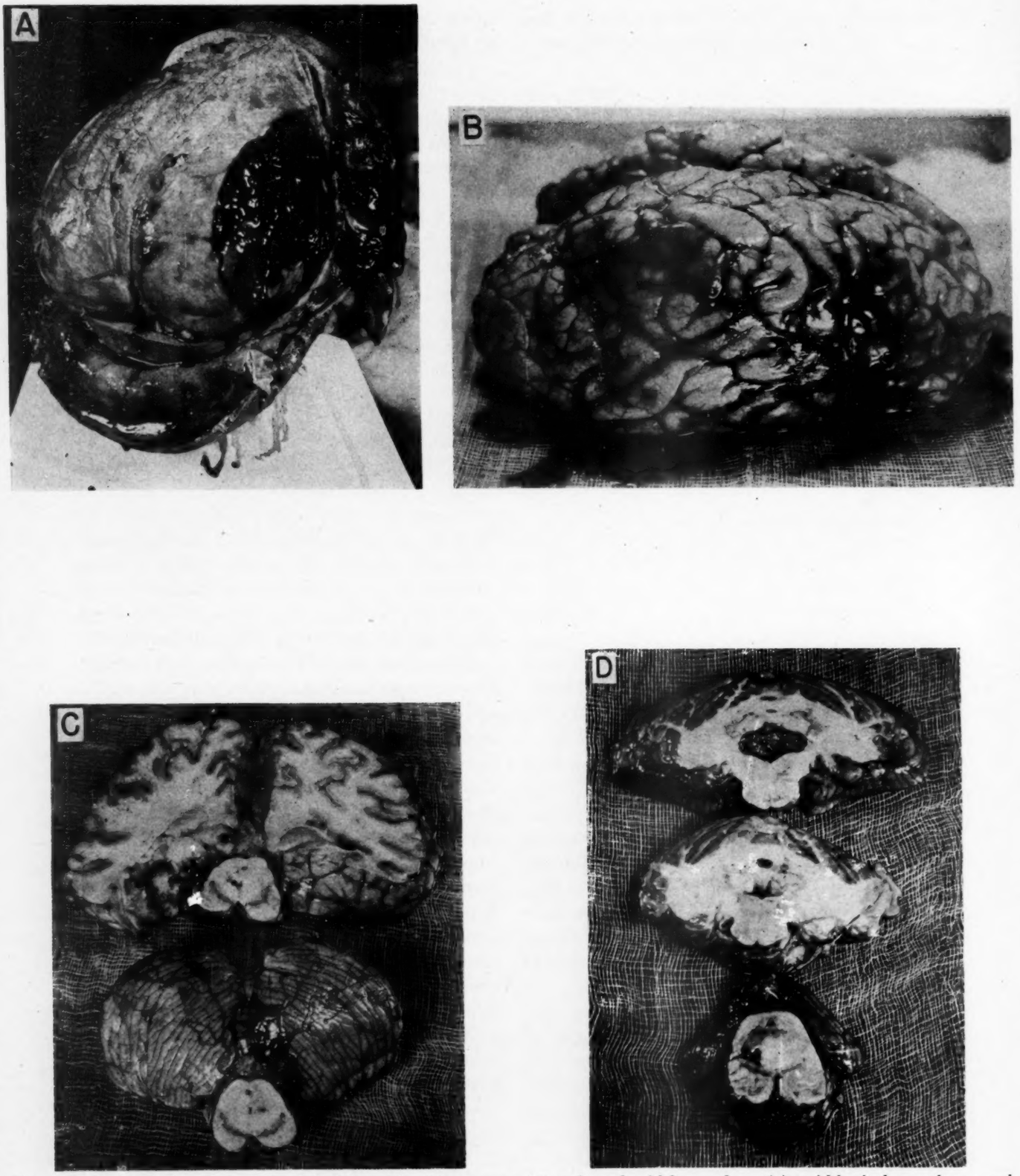

FIG. 1. Autopsy findings in a case with right epidural hematoma (A); subarachnoid hemorrhage (B); mid-brain hemorrhages and contusion of right occipital lobe (C); hemorrhages in the cerebellum and pons (D). That a combination of pathologic conditions co-exist is well known. However, usually a single pathologic entity predominates, producing fairly typical signs and symptoms.

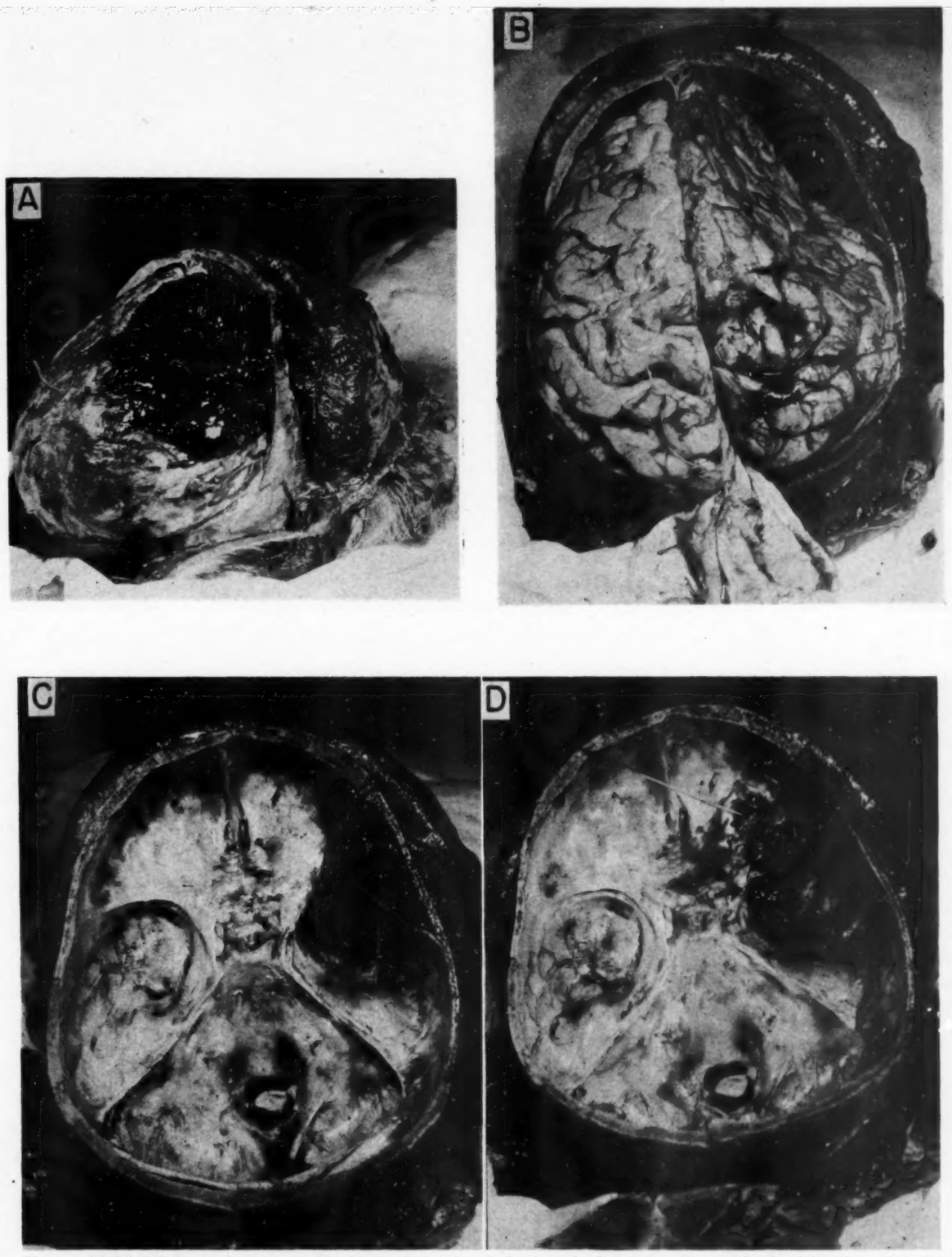

Fig. 2. Epidural hematoma over the convexity (A) causing marked compression of brain (B), dissecting its way into the middle fossa all the way to the region of the superior orbital fissure (C), and dissection showing the extent of the hemorrhage in the superior orbital fissure (D). This explains the usual involvement of the third nerve by compression in its course through the orbital fissure resulting in widely dilated pupil or ocular motor palsy.

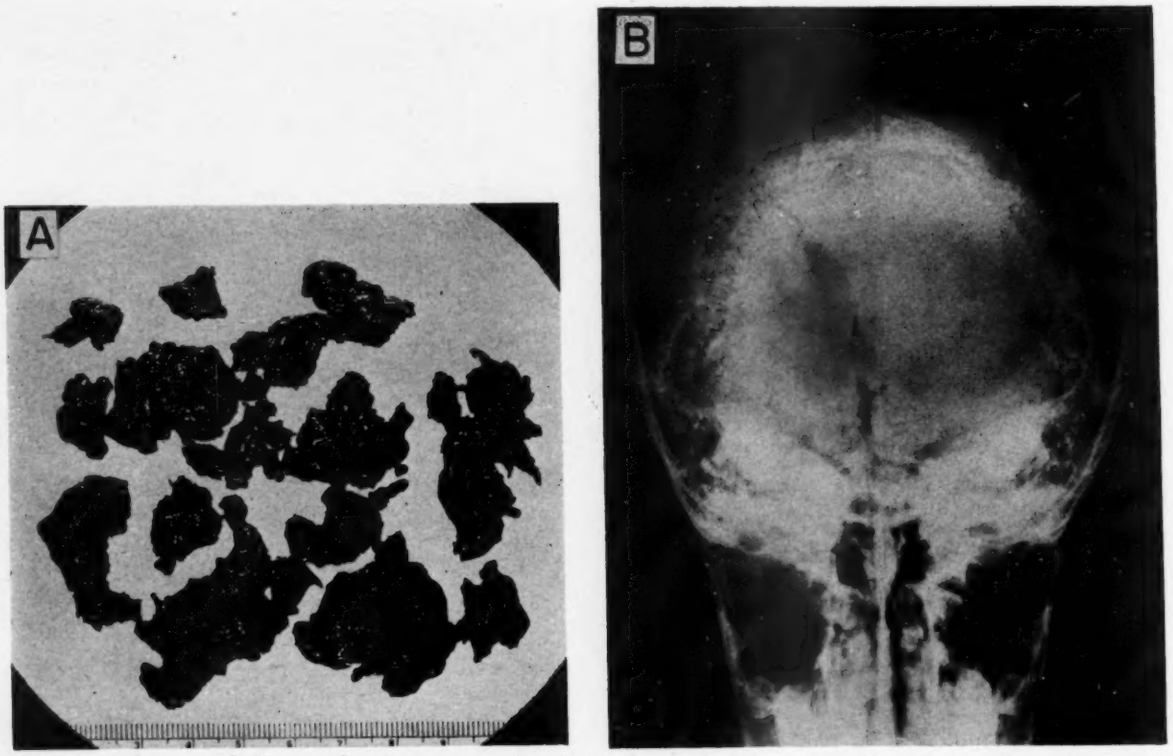

FIG. 3. A and B, encephalogram showing compression of right ventricle from epidural hematoma; hemorrhage only over the convexity with no inequality of pupils.

minor, deserve close observation, particularly if continued unconsciousness and focal signs exist. In two cases, demonstrated by postmortem examination, the fractures were of such degree that roentgen studies could not have revealed them. In one case of bilateral middle meningeal hemorrhage both temporal areas were explored because of the extensive fracture from one temple to the other. The patient presented bilateral neurologic signs. In patients with a pineal calcification, the presence of a shift may be diagnostic and may locate the lesion accurately. (Fig. 7.)

Pathologic Observations. The initial hemorrhage from the middle meningeal vessels may be instantaneous and rapid or delayed and slowly progressive. In progressive lesions, various venous channels of the dura and bone contribute as the separation of the dura from the bone proceeds. Accompanying subdural bleeding was noted in eleven out of thirty cases. In two patients there was an associated subdural accumulation of spinal fluid. In three instances, a massive temporosphenoidal clot was found to accompany the extradural collection.

Treatment. In our experience the operation of choice is that of a subtemporal decompression on the side of the lesion. If the clot is beyond the area of the temporal bone, a small bone flap may be required. Bleeding points are controlled. The middle meningeal artery is ligated or occluded by silver clips or cautery. Deep hemorrhage at the base may require retraction of the temporal lobe for exposure of the foramen spinosum into which a plug of cotton or wood may be introduced. The dura is opened, if necessary, to remove a subdural or intraparenchymatous clot. Usually a gauze drain is used for hemostasis. Local or general anesthesia may be used. In our experience pentothal sodium has been dangerous.

ACUTE SUBDURAL HEMORRHAGE

Acute subdural hemorrhage usually results from a tear of pial vessels associated with contusion and laceration of cerebral

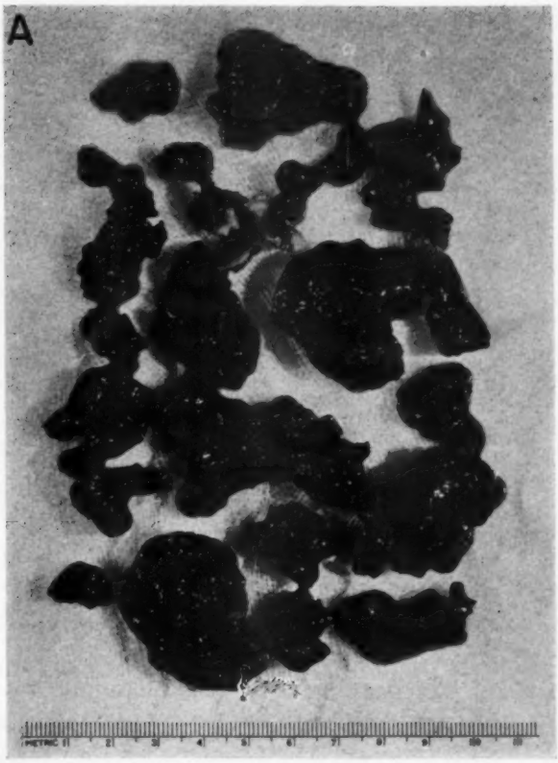

FIG. 4. A and B, epidural hematoma in a patient with a linear fracture of the left parietal bone extending into the middle fossa.

tissue. (Table II.) At times it is extensive, covering the surface of a hemisphere. Bilateral collections may be present. As a rule, the hematoma is located in the fronto-parieto-temporal region of one or both sides. Occasionally an unusual localization

TABLE II
ACUTE SUBDURAL HEMORRHAGE

Lucid interval	8
Continued unconsciousness with disorientation	19
Dilated pupil	17
Hemiparesis or hemiplegia	20
Dilated pupil with contralateral paralysis or paresis	13
Convulsions (Jacksonian)	6
Convulsions (generalized)	4
Total cases	39
Deaths	15

of the clot is seen. (Fig. 5.) In two patients there was an extensive subdural hematoma between the two hemispheres.

The common fronto-parieto-temporal clot site can be explained on the basis of anatomic relations between brain and bone. The frontal and temporal lobes are snugly fitted into a bony encasement having projecting and irregular surfaces. In indirect blows when the rapidly moving head strikes a non-moving or slowly moving object, the intracranial contents by a mass movement abut against the irregular surfaces with resulting vascular disruption in this region.

Clinical Observations. The conscious state may manifest changes which correspond with those observed in patients with extradural hemorrhage. The "lucid interval" is common and is seen as frequently as in middle meningeal hemorrhage. The interval may vary from a few hours to several days or weeks. Since expansion of the subdural lesion occurs more slowly than the epidural, resulting physiologic events are usually less prompt in appearance. The longer the period of lucidity and freedom from major symptoms, the better the prognosis. Patients may remain semiconscious or comatose from the time of injury. Focal neurologic signs may be meager, those present being the result of increased intracranial pressure. Frequent examinations may reveal a gradually deepening stupor with a dulling of responses to stimuli. A slowing of the pulse may occur. Occasionally the blood pressure rises with increasing stupor but as a rule it shows little or no change. Convulsions are common. Pupillary inequality and extraocular palsies occur. A dilated pupil, if present, is usually on the same side as the lesion. Unilateral extraocular muscle paralyses are also usually ipsilateral. There is most commonly an absence of papilledema. Increasing paresis of one-half of the body may be seen. This paresis or paralysis may be on the same side as the subdural collection, resulting in false localization. The spinal fluid pressure is usually elevated but surprisingly low pressures have been noted. The cerebrospinal fluid is often bloody but may be xanthochromatic or clear. Severe bloodly spinal fluid most frequently indicates diffuse intracerebral injury but exceptions to this conclusion are commonly encountered. Failure of a patient who has sustained a cranial injury to improve normally suggests the possibility of an existing complication which may well be a subdural collection. By means of pneumoencephalography or multiple burr holes the diagnosis may be made.

Roentgen Observations. A fracture of the skull may be present on the same or on the side opposite the hematoma with equal frequency. (Fig. 6.) A fracture is commonly absent. The presence of a fracture line does not locate the collection in cases of acute subdural hemorrhage. The presence of a shifted pineal shadow may be diagnostic. (Fig. 7.)

Pathologic Observations. As a rule, the disruption of pial vessels is the source of a subdural hemorrhage. Occasionally a massive intraparenchymatous clot may extrude through the cortex and seep into the subdural space. The latter is more common in non-traumatic cases. Contusion and laceration of the cerebral substance may accompany pial tears; deeper multiple small hemorrhages may be seen. These associated lesions complicate the syndrome of the subdural collection and serve as a cause for

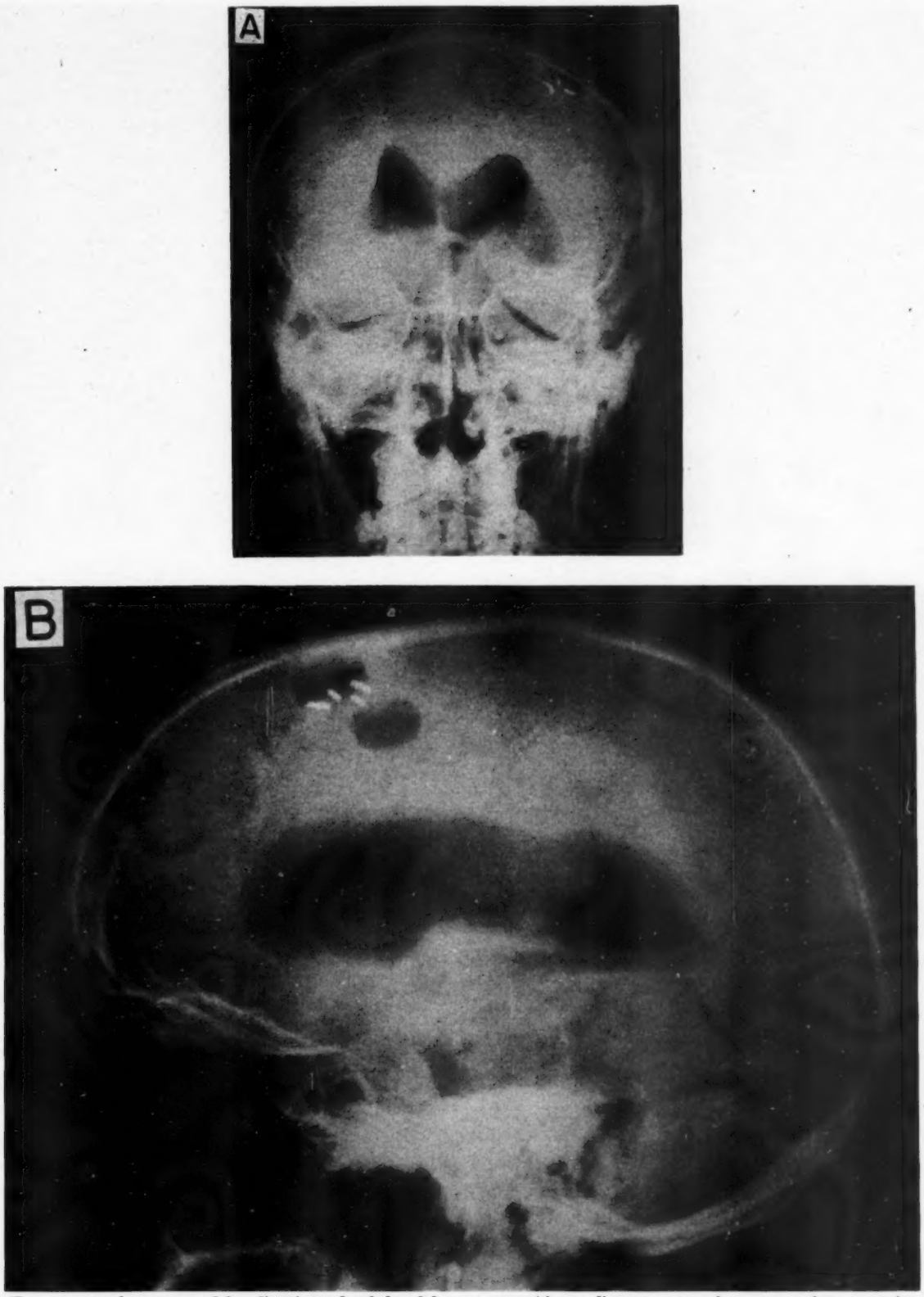

FIG. 5. A and B, unusual localization of subdural hematoma. Air studies suggest a large mass between the two hemispheres which was a subdural hemorrhage from tear of connecting veins between cerebrum and superior sagittal sinus.

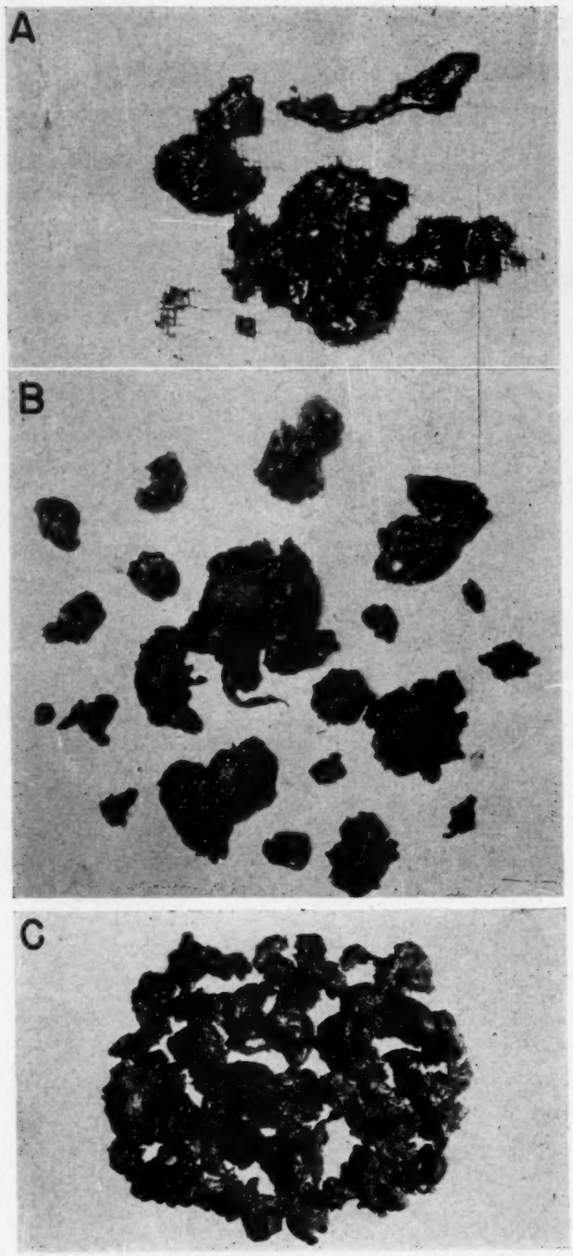

FIG. 6. Subdural hemorrhage (A) in a case with a fracture on the same side as the hemorrhage; (B) in a patient with a fracture on the opposite side; (C) in a patient with no fracture.

prolonged disability or death. A subdural hemorrhage may be of non-surgical proportions producing minor or no symptoms and signs. Upon the initial size and source of the hemorrhage depends the expanding nature of the mass with slow or rapid progression of symptoms and signs. A disruption of a large pial vein may produce practically instantaneous signs and symptoms. In the slowly expanding forms, the absorption of tissue fluids and cerebrospinal fluid by the collection may only gradually add volume and only insidiously result in a dynamic intracranial condition. This process becomes characterized by its chronicity and is so described by the term chronic subdural hematoma. In instances in which the localizing signs are on the wrong side there probably occurs compression of the brain stem on the opposite side from the hemorrhage against the tentorial border, compressing the pyramidal tract on the opposite side.

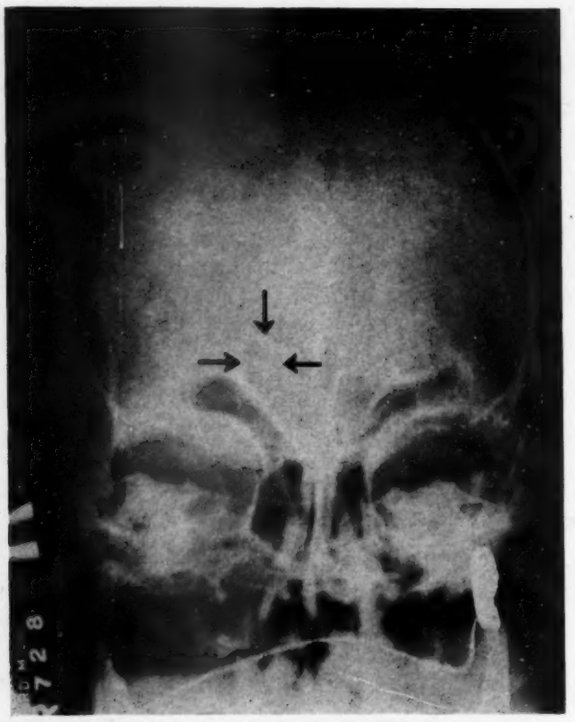

FIG. 7. Pineal shift in a patient with acute subdural hematoma.

Treatment. The acute subdural hemorrhage presenting dynamic signs usually necessitates operative intervention. Both hemispheric surfaces should always be explored. A trephine opening at the frontoparietal junction 2 inches on either side of the midline uncovers the greatest majority of collections. If the two initial openings fail to reveal a hematoma, an air study may be employed in place of further random openings. If a subdural hemorrhage has been located, a subtemporal decompression

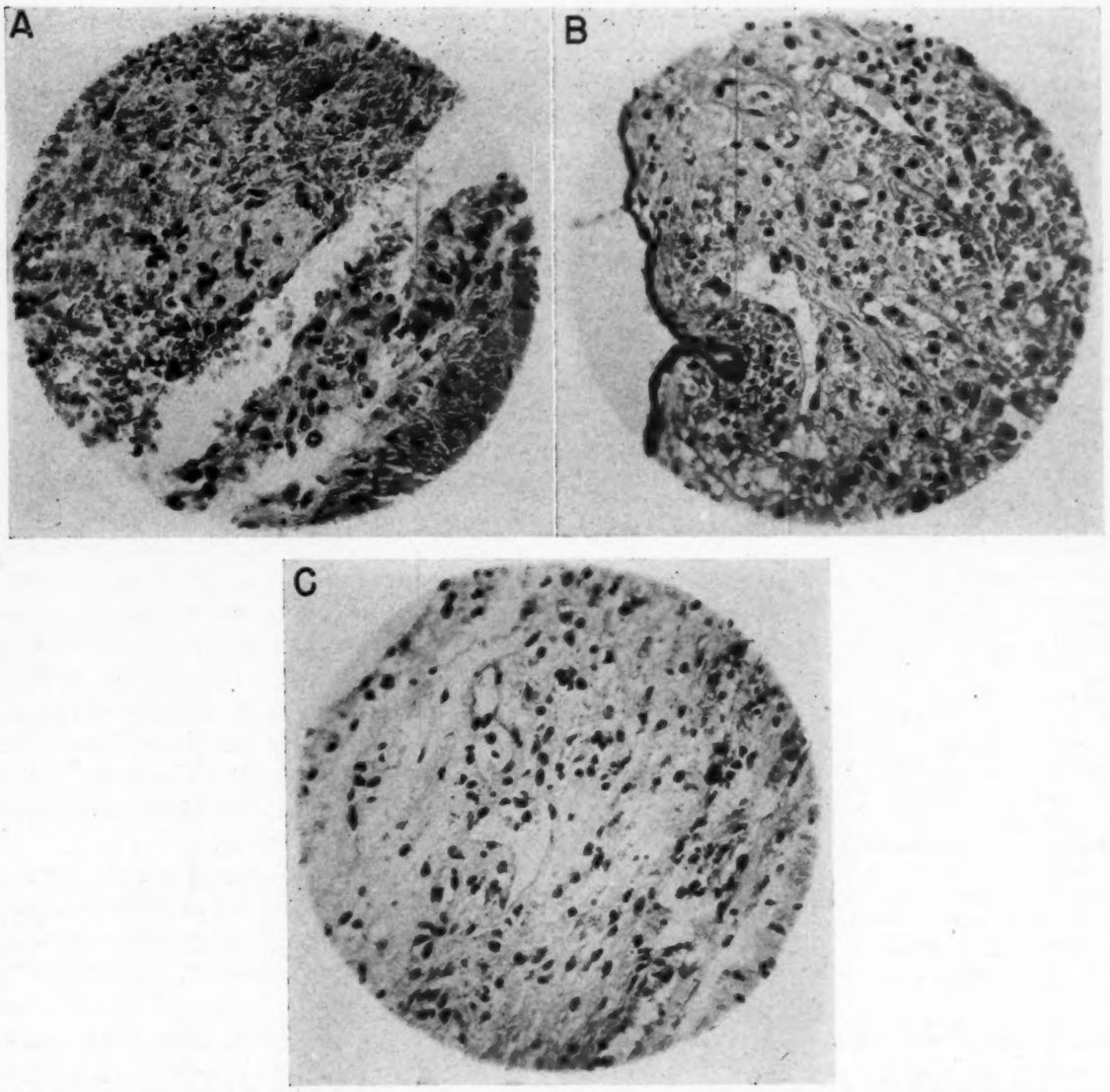

Fig. 8. Connective tissue proliferation in a patient with acute subdural hemorrhage eighteen hours after injury (A). Thick wall on the dural side of chronic subdural hematoma six weeks after injury (B). Thick wall on the dural side of chronic subdural hematoma several months after injury (C).

is then performed on the side of the lesion. The liquid and/or semi-solid clots are irrigated from the subdural space by means of saline and a brain spatula. The subdural space is drained from twelve to eighteen hours.

SUBACUTE AND CHRONIC SUBDURAL HEMATOMAS

When hemorrhage into the subdural space is small in amount or of quantity which is not sufficient to jeopardize the spacial relationships and requirements within the cranial cavity, symptoms may be absent for weeks or months. By osmosis and the diffusion of cerebrospinal fluid and other tissue fluids into the collection (having a higher specific gravity), the subdural hematoma gradually expands. Additional volume is contributed by occasional hemorrhages into the colt from vessels in the proliferating granulation tissue on the dural side of the collection. (Fig. 8.) Ultimately the expansion results in cerebral symptoms. The prolonged, lucid, symptom-free interval can be explained upon these circumstances. (Table III.)

Clinical Observations. Chronic subdural hematoma may follow minor types of cranial injury. In some instances the trauma may be entirely forgotten by the patient and relatives. On the other hand, severe cerebral damage may be accompanied and complicated by this condition. In such cases there may be fracture of the skull with disability from the onset of the injury.

TABLE III
SIXTY-FIVE OPERATED CASES OF CHRONIC SUBDURAL HEMATOMA (ADULT)

Headaches	
Present over the lesion	42
Not complained of, or no history	12
Generalized	11
Pupils	
Dilated on side of lesion	13
Equal	49
Dilated on side opposite lesion	3
Extraocular palsy	
Third	4
None	61
Fracture of skull	
Present	21
None	44
Side involved	
Left	32
Right	27
Bilateral	6
Spinal fluid findings	
Pressure above 300	40
200–300	12
100–200	5
Blood or xanthochromatic	
Operation of choice:	
Bilateral, exploratory, trephine subdural temporal decompression on the side of lesion or osteoplastic flap if clot is solid	
Recovered	59
Died	6

If chronic subdural hematoma complicates a severe injury, the patient may remain unconscious or semi-conscious for varying periods from days to weeks, progressing from the unconsciousness caused by the initial severe cerebral damage into a semi-consciousness caused by the cerebral compression of the subdural hematoma. A number of patients in this series had associated severe, diffuse cerebral injury.

In the more typical case the patient may be normal following a cranial injury for varying periods. Then in a brief period of but several days headache, previously of mild order, becomes intense. Conduct and personality defects appear and within a week the patient progresses into a stupor, and finally coma if recognition of this complication has not been made.

Pupillary inequality may occur with the enlarged pupil on the ipsilateral side. The fundi may exhibit papilledema although this is the exception rather than the rule. Visual field studies in those patients who are cooperative have proved of little help. Headache is a constant complaint and may be localized to the side of the lesion. An overlying hyperpathic zone of the scalp may be present. In some patients increasing drowsiness may be the only finding. As a general rule, focal neurologic signs obtain in the advanced cases. Bilateral focal signs and findings implicating the "wrong side" are common. This is the result of peduncular compression against the tentorium on the side opposite the lesion. The cerebrospinal fluid pressure is usually elevated. The fluid itself may be clear but is frequently xanthochromatic. The grossly clear fluid may contain an elevated total protein with minimal pleocytosis.

Changes in pulse, respiration, blood pressure and temperature are of little diagnostic significance until advanced cerebral compression has occurred. At times a bradycardia may be observed usually accompanied by headache. The electroencephalogram may be of important diagnostic assistance. (Figs. 9 and 11.)

Roentgen Observations. A fracture of the skull may or may not be present and its presence does not indicate the side of the hematoma. As a rule, the complication follows minor trauma to the head and thus a majority of patients show no fracture. Pneumoencephalograms are very helpful and patients with subdural hematomas tolerate this procedure surprisingly well. If the ventricles are visualized, there is a midline shift and cortical air markings on the affected side are characteristically absent. (Figs. 9 and 10.) A collection of air may show medial to the hematoma and this column may appear as a "pointer" within the cranial cavity. This finding is pathog-

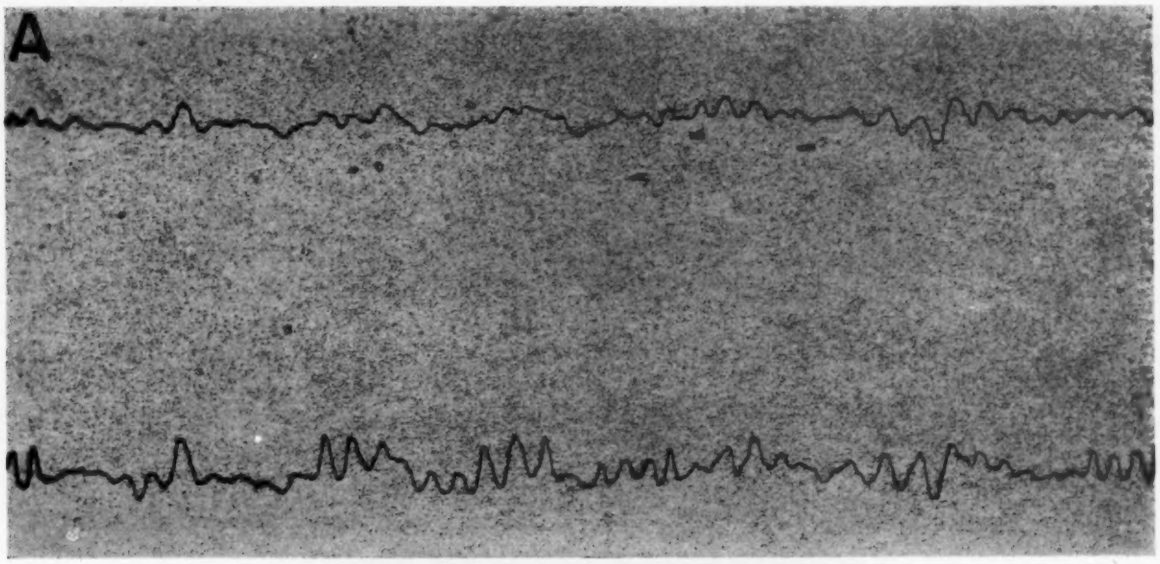

FIG. 9. A and B, air study and electroencephalogram in a patient with right subdural hematoma. Note absence of subarachnoid spaces on the affected side. Electroencephalographic record with disorganization of the electrical pattern on the affected side.

nomonic of a subdural hematoma. Pineal shadow shift may be diagnostic of the location of the lesion. (Fig. 7.) Electroencephalographic studies may be diagnostic (Fig. 11), although in our experience correct localization of the lesion has been accomplished by this technic in about 40 per cent of the cases.

Pathologic Observations. In the genesis of chronic subdural hematoma there is a prompt cellular reaction with connective tissue proliferation on the dural side of the collection. (Fig. 8.) On the arachnoid side a limiting membrane forms. The subdural mass increases in size by the absorption of tissue fluids and spinal fluid through the process of osmosis and diffusion. The inner layer of the sac serves as a semi-permeable membrane. Thus an expanding process occurs (usually covering a wide area of the

FIG. 10. Marked compression of the brain with ventricular distortion in a case of acute subdural hematoma.

cerebral surface, since the subdural space is not limited) rather than an absorption of the collection. Within the sac of the hematoma the blood may partially or completely hemolyze. It is not unusual, however, for the clot to remain semi-solid.

Treatment. Bilateral trepanation at the frontoparietal junction about 2 inches on either side of the midline followed by a subtemporal decompression on the side of the collection is the method of choice if the lesion is mostly fluid. A small osteoplastic flap is used if the clot is mostly solid. A single or two trephine openings may be adequate for the evacuation of unclotted hematomas when combined with irrigation. Changing the position of the head may also be of assistance. It is now generally admitted that the membrane of the hematoma sac need not be removed. After evacuation of the sac contents the ability of this tissue to retain its characteristics of a semi-permeable membrane is lost.

SUBDURAL HEMATOMA (ACUTE AND CHRONIC) IN INFANTS

Newborn infants occasionally are afflicted with subdural hemorrhage. Birth injuries, injury by forceps, falls and other etiologic factors may be responsible. In acute subdural hemorrhage, the infant does not react normally to its environment, presenting often a lethargy and dulled response to stimuli. A weak, inconstant cry replaces the lusty ability of the normal infant. Cyanosis appears with weak, irregular respirations. High concentrations of oxygen may have little effect. Neurologic findings may be of a minor order. Occasionally spasticity is present on the contralateral side. Pupillary irregularities occur. Convulsions are common, being either localized or generalized. An important finding is a tenseness, fullness or bogginess of the anterior fontanel. Lumbar puncture usually shows a grossly bloody fluid under increased pressure. .

The infant with a chronic type of subdural hemorrhage or hydroma has progressed satisfactorily in its early months until a parent or pediatrician notices enlargement of the head. In other patients, a convulsion first draws attention to an interruption of normal progress. In all other respects, the growth, sleep, feeding, vision, etc., may be normal. Head measurements indicate an enlargement above normal. A cracked pot sound may be noted on percussion of the head. The fontanel is wide and the sutures may be separated. Roentgen studies may show this same suture separation. Focal neurologic signs and fundus changes are usually absent.

The lesion may be accurately diagnosed by tap with a No. 18 gauge needle through the lateral aspect of the fontanel on both sides. When present, the collection may be evacuated through appropriate trephine openings on the affected side. Occasionally aspiration alone has been sufficient to drain the collection.

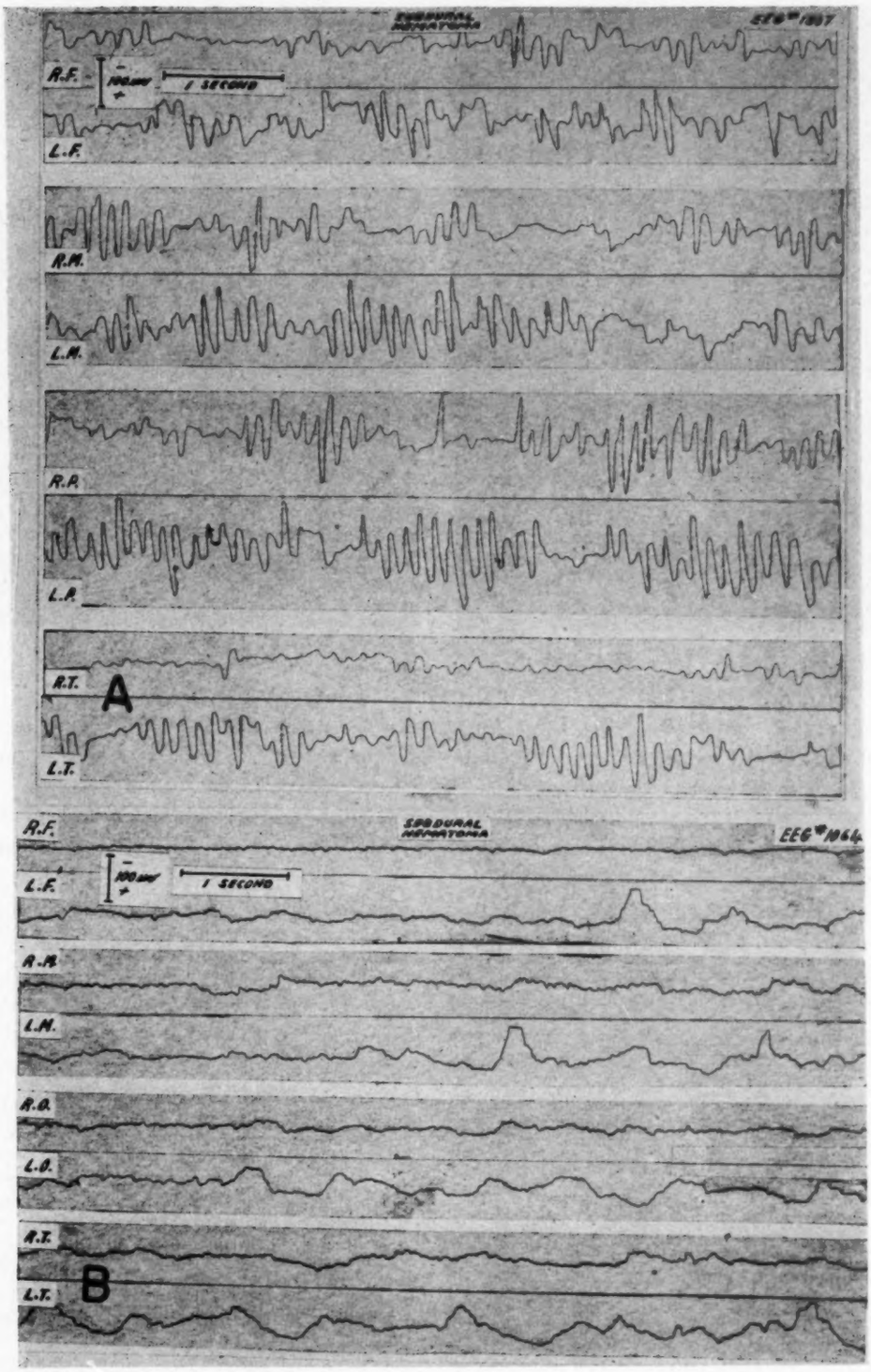

FIG. 11. Electroencephalographic records in two cases of subdural hematoma. In (A) there is disorganization of the electrical pattern over the right temporal region. In (B) there is delta activity over the left hemisphere.

FIG. 12. A and B, subdural hematoma in a four months old infant visualized by injecting air after drainage of fluid contents. Note that the entire hemisphere was compressed by the fluid mass.

The chronic subdural collection is suitably managed by means of two frontoparietal openings of an exploratory type. If a clot is encountered with a thick membrane, it may be necessary by means of a small bone flap to remove this layer in order to allow expansion of the depressed hemisphere. (Fig. 12.) Clots may be irrigated from the surface through two openings. A hydroma may be evacuated through a single opening.

SUBDURAL ACCUMULATION OF SPINAL FLUID

Consideration must be given to the occurrence of a subdural accumulation of spinal fluid. (Table IV.) This curious condition is seen in less severe grades of head injury. It is almost never diagnosed prior to operation. That there is a cause and effect relationship between this condition and clinical findings is proved by the rather remarkable results following treatment in some cases. Pathologically, the collection has been stated to be caused by a rent in the arachnoid with a resulting valve-like action which permits cerebrospinal fluid to enter the subdural space and not return. When a patient fails to improve after drainage of a collection, air studies may be necessary to rule out a co-existent disturbance. We have seen middle meningeal hemorrhage occur with this lesion. In some cases this abnormality may be seen without a history of cranial injury.

TABLE IV
SUBDURAL COLLECTION OF SPINAL FLUID

Lucid interval	3
Continued unconsciousness with disorientation	16
Dilated pupil	9
Hemiparesis or hemiplegia	8
Dilated pupil with contralateral paralysis or paresis	5
Convulsions (Jacksonian)	2
Convulsions (generalized)	2
Total Cases	29
Deaths	7

The treatment is that of simple drainage of the subdural collection through trephine openings. Not infrequently the collections are bilateral.

MASSIVE TYPE INTRAPARENCHYMATOUS HEMORRHAGE

Massive intraparenchymatous hemorrhage or intracerebral hemorrhage in acute head injury is uncommon in autopsy ma-

terial. The most vulnerable area seems to be in the region of the temporosphenoidal lobe. Clinically the same rarity of such cases is evident. In our series of over 200 operations for head injury with massive hemorrhage, there have been five instances of massive intracerebral clot; three occurred in the left temporosphenoidal lobe.

The clinical findings observed were similar in each instance to left temporosphenoidal clot. A progressive failure characterized by a right lower facial weakness, right upper limb paresis, eventual aphasia and stupor was noted. Because of the localizing signs, trephine with a left subtemporal decompression was performed and in each instance a massive clot from the temporosphenoidal lobe with an overlying subdural hemorrhage was evacuated. Much necrotic brain tissue was encountered which was also removed. (Fig. 13.)

In the diagnosis of such cases, localizing neurologic signs aided by air studies should accurately locate the lesion.

FIG. 13. Extensive contusion of brain in right parietal region. Such a lesion may eventually result in softening and hemorrhages causing an intraparenchymatous mass lesion. Traumatic intraparenchymatous hemorrhage is almost always associated with softening and necrosis of brain tissue resulting in severe cerebral deficit.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage is the most common variety of traumatic intracranial hemorrhage. It may co-exist with epidural, subdural and intraparenchymatous hemorrhage. The associated subarachnoid hemorrhage is usually brought about by laceration and contusions of brain surfaces.

TABLE V*

Dilated pupil	11
Hemiparesis or hemiplegia	15
Lucid interval	7
Continued unconsciousness or disorientation	26
Dilated pupil with contralateral paralysis	10
Convulsions (Jacksonian)	4
Convulsions (generalized)	3
Total cases	34
Deaths	16

* In thirty-four cases the above signs and symptoms led to exploration, but the latter revealed no massive clots. Usually edema of the brain, contusions and subarachnoid hemorrhage were seen.

At times it is extensive enough to cover one or both hemispheres. The diagnosis is confirmed by a lumbar puncture. The cerebrospinal fluid obtained from the spine may contain much less blood than that obtained from the cisterna magna or the ventricles. Localized subarachnoid hemorrhage with edema may cause focal signs of a progressive nature so as to resemble closely the picture of subdural hematoma or massive intracranial hemorrhage. The presence of blood in the spinal fluid should not deter operative intervention, for in cases of epidural and subdural hemorrhage there is frequently associated subarachnoid hemorrhage. The patient with subarachnoid hemorrhage usually gradually improves, the conscious or semi-conscious state lightens and focal signs resolve. Conservative supportive treatment is thus wisely continued in the presence of gradual improvement.

PETECHIAL HEMORRHAGE

Brief mention of petechial hemorrhages is made to complete the subject. How often they occur in those with severe cranial injury is difficult to establish, but 34 per cent of cases seen at the autopsy table show these small hemorrhages throughout the cerebral tissue. The association of petechial hemorrhages with other forms of operative

conditions makes for longer convalescence and less complete recovery due to sequelae of a permanent nature.

COMMENT AND SUMMARY

A similarity of symptoms and signs is evident in the discussion of the various types of acute traumatic intracranial hemorrhagic collections. The state of consciousness and its alterations are significantly important. An increasing stupor, abolition of the conscious state after a lucid interval are indications for possible operative intervention. The neurologic status of the patient should be studied frequently. The progress of neurologic findings may be suggestive of an enlarging mass lesion. On repeated examinations, if the patient shows increasing weakness of one-half of the body, eventuating in paralysis, he may be afflicted with an enlarging blood clot. On the other hand, if a paresis or paralysis has been found to exist soon after injury, its causation by contusions and bruises of cerebral tissue is much more likely and, therefore, operative intervention may not be considered in such a case. The presence of inequality of pupils may be significant. The dilated pupil is usually on the side of the lesion. Papillidema may be present although this is rare. The vital functions may show alterations from normal. A slowing pulse rate and a lowering respiratory rate may accompany mass lesions. An increase in blood pressure, although rare, may signify increasing intracranial tension.

In acute hemorrhagic collection the one single important symptom justifying exploration is increasing drowsiness or stupor. Pupillary inequalities, changes in vital functions and focal neurologic signs may corroborate the presence of an enlarging clot. However, a dilated pupil, a low pulse rate or hemiplegia in a perfectly conscious patient does not justify operative intervention.

In subacute and chronic subdural hematomas the presence of unilateral headache and hyperpathia of the scalp over the pathologic lesion are important. The association of severe brain injury with subdural hematoma produces long disability and the recognition of a complicating subdural hematoma in long continued unconsciousness is important.

Post-traumatic epilepsy is rare in epidural and chronic subdural hematomas. In acute subdural hematomas it is seen in almost a third of the patients. This favors the assumption that in these patients the subdural hematoma is associated with contusions and bruises of the cerebral surface eventuating in cerebrodural cicatrization. Headaches, dizzy spells and personality changes are surprisingly uncommon as sequelae in postoperative mass lesions.

SURGICAL TREATMENT OF TUMORS OF THE PITUITARY BODY*

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THE pituitary tumors observed by the senior author during a period of approximately fifteen years provide the material for this report. Histologic verification was not obtained in every case, a fairly large proportion of patients having been treated by means of irradiation exclusively. In the non-verified cases, the combination of clinical symptomatology and physical findings, together with the results of visual field studies and roentgen examination of the skull, established the diagnosis beyond reasonable doubt. Whenever the nature of the lesion was questionable, the issue was settled by operation. A few patients were seen only once or twice in consultation but sufficient pertinent data concerning them were made available to warrant including them in this study.

CHROMOPHOBE ADENOMAS

The chromophobe adenomas constituted the largest group of tumors, there being ninety-three cases in this series. The incidence of males was somewhat higher than that of females, the figures being fifty-two and forty-one, respectively. That the disease is primarily one of adults is indicated by the fact that 74 per cent of the patients were in the fourth, fifth or sixth decade of life at the time they sought medical attention. The fifth decade accounted for 34 per cent of all patients, the largest number in any comparable age group. Three patients were less than twenty years old.

Symptomatology. The most constant complaint was impairment of vision. It was either the chief symptom or one of the presenting complaints in eighty-six cases. Next in frequency were headaches which occurred in fifty-six patients. Disturbances referable to sexual function, amenorrhea or, in the male, loss of libido and potency, were present in fifty patients. Not infrequently the amenorrhea or impotence antedated the visual symptoms. Less often mentioned in the anamnesis were increasing obesity, a tendency toward somnolence, generalized weakness, diplopia and, in a few cases, polydipsia and polyuria. Mental symptoms were pronounced in six instances. Two of these patients presented unusual clinical pictures and in each case the diagnosis was resolved by pneumencephalographic examination, which revealed the tumor to be above the sella turcica instead of chiefly within it and projecting into the third ventricle. In both cases, the nature of the lesion was subsequently verified. A third patient proved to have a large intracranial extension into the middle fossa on one side. Mental symptoms and convulsions occurred in still another patient who had been operated upon twice previously, once twenty years ago and again thirteen years ago. Following the evacuation of a cystic tumor at a third operation, he improved considerably. He is still alive and continues to maintain his improvement. In a fifth case, recurrent growth of a tumor was associated with a manic state which disappeared after operation. This patient has been followed for a period of fourteen years and has remained free of any further recurrence. The remaining case was one in which headache, drowsiness and confusion supervened following a relatively minor head injury. A subtotal removal of the tumor was accomplished at operation but the patient expired postoper-

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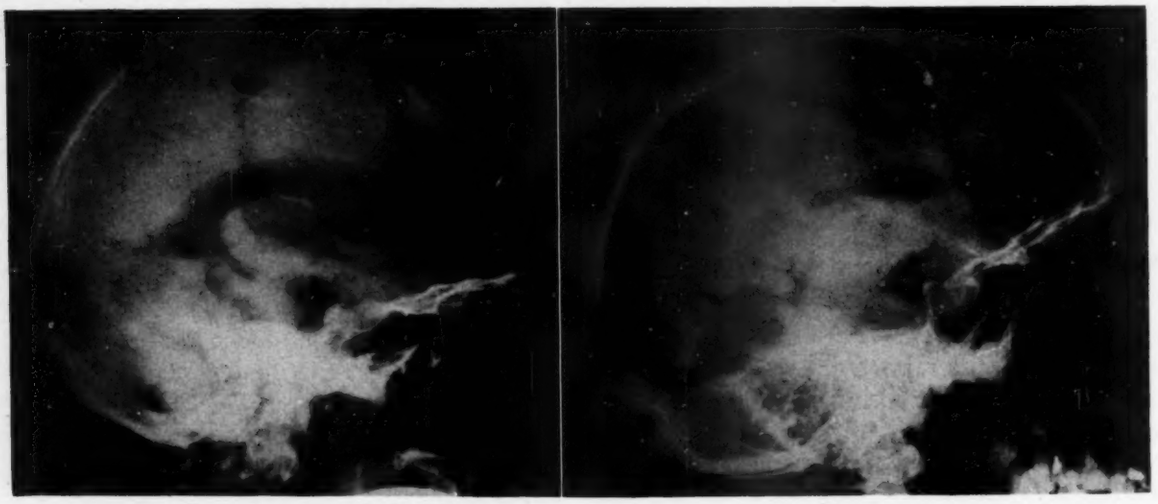

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FIG. 1. Case 90. Pneumencephalogram following recurrence of symptoms and identical with original preoperative pneumencephalogram. Obliteration of anterior portion of cisterna interpeduncularis with concave defect rostrally.

FIG. 2. Case 67. Failure of visualization of the cisterna chiasmatis and of the cephalic half of the cisterna interpeduncularis. Note normal-sized sella turcica.

atively. A necropsy not having been performed, the extent of the lesion remains unverified.

The objective evidence supporting the diagnosis of a chromophobe adenoma was of a dual character. It consisted of constitutional stigmas resulting from diminished activity of the hypophysis, plus signs representing local pressure effects of the enlarging mass. Disturbance of sexual function, a cardinal manifestation of hypopituitarism, has already been mentioned and the frequency of its occurrence in this series noted. In fifty-four cases one or more of the following additional physical features were recorded: an unusual degree of pallor often described as a "waxy" or "pasty" complexion, dryness and atrophy of the skin, sparseness of the beard and a female escutcheon in males, a paucity of axillary and pubic hair, muscular flabbiness, obesity, and in men, a feminine type of fat distribution. Blood sugar levels were usually normal and sugar tolerance tests also within normal limits. The basal metabolic rate was almost uniformly diminished, averaging −14 per cent. Roentgenographic evidence of an intrasellar tumor was present in all but three cases. The characteristic findings included enlargement of the sella turcica with variable degrees of erosion of the posterior clinoids, dorsum sellae and sellar floor.

One of the three cases in which the sella turcica was normal in size showed atrophy of the posterior clinoid processes and dorsum sellae. All three patients were further studied by means of pneumencephalography performed through the lumbar route. Obliteration of the anterior portion of the cisterna interpeduncularis together with a concave defect rostrally was demonstrated in one case. (Fig. 1.) Failure to visualize the cisterna chiasmatis and the cephalic half of the cisterna interpeduncularis occurred in another patient. (Fig. 2.) Deformity of the cisterna interpeduncularis by a mass which also projected into the third ventricle was revealed in a third encephalogram. (Fig. 3.) Ventriculography was employed in two cases that presented diagnostic problems. The sella turcica was enlarged and deformed in both but the clinical pictures were most unusual for a pituitary adenoma. The ventriculograms of one of these patients revealed a mass arising from the region of the chiasm which protruded into the cisterna interpedun-

cularis and also elevated the anterior aspect of the third ventricle. On laminagraphic section it was possible to demonstrate a large tumor eroding the anterior wall and floor of the sella and filling the sphenoidal sinus. (Fig. 4.) In the second case, the films showed considerable dilatation of the lateral ventricles and a deformity of the anterior portion of the third ventricle outlining a tumor mass. Concave filling defects in the floors of the frontal horns and an elevation of the cisterna interpeduncularis were also demonstrable. (Fig. 5.) The diagnosis of a chromophobe adenoma was subsequently verified histologically in all five cases studied by pneumencephalography.

Table I

Type of Defect	No. of Cases
Bitemporal hemianopsia	63
One eye blind or barely perceiving light; temporal field defect in the other eye	14
Homonymous hemianopsia	6
Bilateral blindness	1
Bilateral light perception	1
Unilateral temporal field defect	2
Light perception in one eye; other eye normal	1
Inferior altitudinal hemianopsia	1
No defect demonstrable	2
Total	91

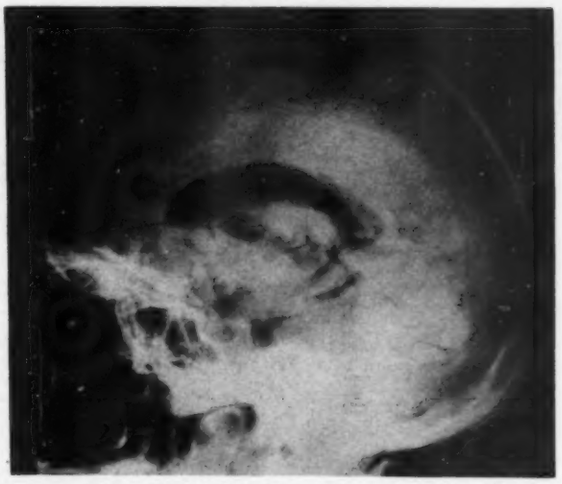


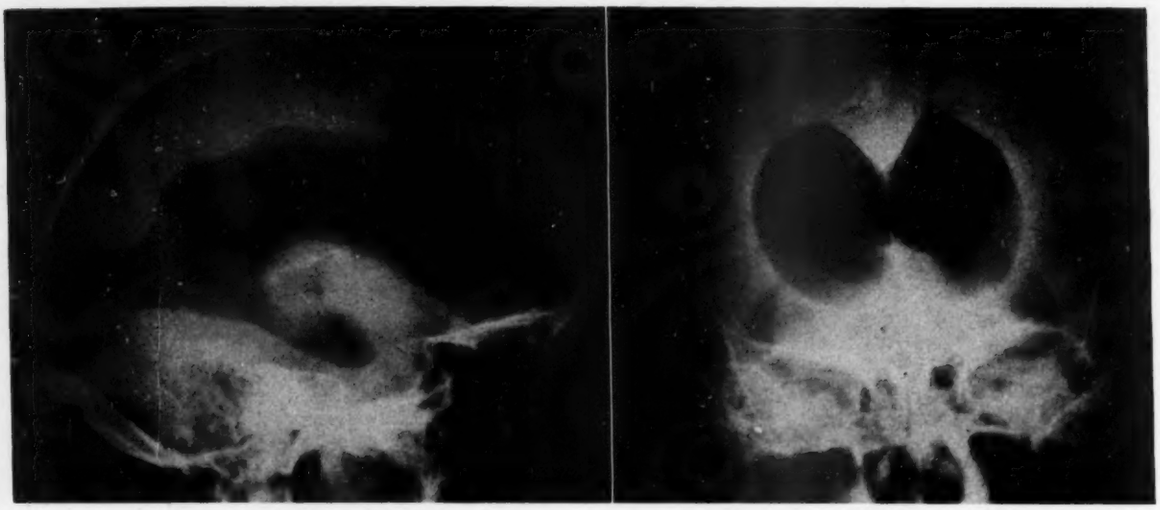
Fig. 3. Case 87. Deformity of cisterna interpeduncularis by a mass also projecting into third ventricle.

Examination of the fundi disclosed optic atrophy in seventy-nine patients. Both discs were atrophic in sixty-two patients while in the other seventeen unilateral funduscopic changes were observed. Papilledema was found in only one case but was reported to have been present in another patient prior to his original operation elsewhere. The various types of visual field defects observed and their incidence is recorded in Table I.

It was impossible to chart the fields in one case owing to the torpid state of the patient. In another case, the nature of a visual defect which subsequently receded after therapy was not established. Studies of the patients in whom a unilateral tem-

A B

Fig. 4. Case 77. A and B, pneumencephalographic evidence of a mass arising from region of chiasm, projecting into cisterna interpeduncularis and elevating anterior portion of third ventricle is not well demonstrated in the photographs. Advantage of a laminagram is clearly indicated. On the plain films, there is pooor visualization of the sella turcica. Laminagram distinctly reveals enlargement of the sella and erosion of the anterior wall and floor.



A B

FIG. 5. Case 86. A and B, dilated lateral ventricles and a deformity of the anterior portion of the third ventricle outlining the tumor. There are also concave filling defects in the floors of the frontal horns and an elevation of the cisterna interpeduncularis.

poral field defect was initially discovered revealed a temporal defect in the other eye during the subsequent course of the illness in one and a disappearance of the defect following therapy in the other. On reexamining postoperatively the patient with light perception in one eye and a normal field in the other, an inferior altitudinal hemianopsia was demonstrated. Examination at still a later date revealed further recession of the scotoma and an inferior nasal quadrantic defect remained. The inferior altitudinal hemianopsia which was observed in another patient was most marked in the right field of vision. At operation the tumor was seen to compress the right optic nerve against the optic foramen. This observation, in all probability, accounted for the bizarre visual field.

Extraocular muscle palsies and disturbances referable to the trigeminal nerves were recorded in a few instances. Eight patients complained of diplopia; in four, there were manifest extraocular muscle paralyses. Unilateral ptosis was present in an additional two cases. Trigeminal involvement, both motor and sensory, combined with an ipsilateral ophthalmoplegia was observed in a totally amaurotic patient. Pain in the distribution of the supraorbital nerve was the chief complaint in another instance though objective evidence of sensory deficit was lacking.

Hemiparesis occurred late in the course of three cases in all of whom the tumor had extended intracranially into the middle fossa. It was associated with Jacksonian seizures in one and with a sensory and motor aphasia in another. Unilateral upper motor neuron signs were elicited in still another patient in whom the tumor had grown into the third ventricle and blocked the foramen of Monro. Convulsions occurred in an advanced case which at necropsy revealed extension of the growth into the third ventricle and the left temporal lobe.

Treatment. It is the practice in this clinic to treat pituitary tumors initially by means of irradiation unless reasons exist which make surgical intervention imperative, chief among which is a severe impairment of vision that makes the danger of amaurosis an imminent one. Most cases in this series were treated in accordance with this view. Careful surveillance of the patient's progress was maintained during radiation therapy, the all important guides being the direction of change of the visual acuity and visual fields. Wherever visual impairment was of a degree to cause concern, treatment was instituted in the

hospital where close cooperation was maintained with the radiologic and ophthalmologic departments. In these cases, visual fields and acuity were examined at least once a day and more often if thought necessary. Provided further loss of vision was halted, even if no improvement was apparent, radiation therapy was continued. If, on the other hand, vision measured in terms of acuity and visual fields deteriorated further, operation was promptly undertaken. Cases favorably affected by irradiation received several such courses of treatment. The technic employed is that described in the monograph of Dyke and Davidoff,[8] the physical factors being: 200 KV constant potential machine, 50 cm. target skin distance, filter of ½ to 1 mm. Cu and 1 mm. Al, and a current of 8 ma. Three portals were used, a right and left temporal, 8 cm. in diameter, and a central forehead area, 5 to 6 cm. in diameter. Treatment was administered daily or every other day and consisted of 200 r to one portal per day, rotating the several portals in sequence. The total amount of radiation per portal varied between 800 r and 1000 r, so that a complete course consisted of 2,400 to 3,000 r measured in air without backscattering. Usually three or four such courses of treatment were prescribed, spaced at intervals of four to eight weeks, depending on the degree of visual disturbance. Inasmuch as the patients in this series were not treated by any one radiologist, the plan of therapy just outlined was not rigorously adhered to in every case. In general, however, it is a schematic representation of the treatment administered to most patients subjected to radiation in our series.

There were three indications for operation: (1) an advanced degree of visual loss bordering on amaurosis; (2) progressive visual impairment despite adequate irradiation or an arrest of visual failure at an unsatisfactory level; (3) a clinical picture not altogether typical of a pituitary tumor and compatible with another type of chiasmal lesion.

Operation was performed in all cases through a transfrontal approach. A coronal skin incision has in recent years largely replaced the Cushing and Frazier types of flap except in the case of male patients who are bald or whose hairline is receding. It is customary in this clinic to operate on the side of the non-dominant hemisphere regardless of whether or not this happens to be the side of the more seriously affected eye. The chiasmal region is reached through an intradural approach, reserving the extradural route, in which the dura is opened along the sphenoid ridge, for cases undergoing secondary operations.

Table II
RADIATION THERAPY WITH IMPROVEMENT

Cases	Degree of Improvement	Duration of Follow-up
1. F. Y. (M. H. #42294)	Slight	5 mo.
2. B. L. (J. H. #251856)*	Marked	5 yr.
3. M. S. (M. H. #41698)	Marked	11 mo.
4. H. R. (J. H. #243257)	Marked	6 yr.
5. P. J. (non-hospitalized case)	Slight	10 yr.
6. M. K. (non-hospitalized case)	Slight	8 yr.
7. A. C. (non-hospitalized case)	Marked	2 yr.
8. A. M. (non-hospitalized case)	Marked	2½ yr.
9. E. S. (non-hospitalized case)	Marked	1½ yr.
10. B. S. (non-hospitalized case)	Marked	5½ yr.
11. H. C. (non-hospitalized case)	Marked	7½ yr.
12. J. B. (non-hospitalized case)	Marked	1 yr.
13. R. H. (non-hospitalized case)	Marked	4 yr.
14. A. F. (non-hospitalized case)	Marked	11 mo.
15. M. B. (non-hospitalized case)†	Marked	17 mo.
16. B. B. (non-hospitalized case)‡	Marked	3 yr.
17. E. B. (non-hospitalized case)	Marked	1½ yr.

* Return of menses; had baby.

† Relief of headaches; normal visual acuity and visual fields prior to institution of therapy.

‡ Relief of headache; retention of good vision existing prior to therapy.

In ninety of the ninety-three cases, adequate data exist to permit analysis of the results of treatment. These are listed in Tables II through VIII.

Fifty-nine patients were treated initially with irradiation. Improvement resulted in seventeen patients, 29 per cent. (Table II.) In another seven cases, 12 per cent, there was improvement with subsequent regres-

TABLE III

RADIATION THERAPY—IMPROVEMENT WITH SUBSEQUENT RECURRENCE

Cases	Duration of Improvement	Subsequent Treatment	Subsequent Course
18. B. B. (J. H. #263557)	Marked improvement; regression after 3½ yr.	Further x-ray therapy	Receiving treatment
19. I. S. (J. H. #265103)	Marked improvement; regression after 6 mo.	Further x-ray therapy failed to halt decline of vision; operation	Marked improvement; 3½ yr. follow-up
20. A. C. (J. H. #260561)	Slight improvement; regression after 3 yr.	Operation	Postoperative death
21. L. W. (non-hospitalized case)	Marked improvement; regression after 5 yr.	Further x-ray therapy	Improvement; 7 yr. follow-up
22. M. B. (J. H. #254458)	Slight improvement; regression after 20 yr. (rapidly became amaurotic)	Further x-ray therapy	Relief of headache; no return of vision; died 2 yr. later; details unknown
23. B. K. (N. I. #30134) Mixed tumor	Improvement; regression after 4 yr.	Further x-ray therapy; progressive mental symptoms; operation 4 yr. later	Died 3 mo. after operation
24. R. H. (J. H. #285908) Cystic adenoma.	Slight improvement 8 mo., recurrence of headache	Operation	Marked improvement; recurrence headache and visual loss 4 yr. later; marked improvement with x-ray therapy; recurrence 3 yr. later; transient improvement with x-ray therapy; reoperated; slight improvement, 1½ yr follow-up since second operation

TABLE IV

RADIATION THERAPY—UNIMPROVED—VISION NO WORSE

Case	Follow-up	Additional Data
25. I. F. (non-hospitalized case)	17 mo.	
26. D. S. (non-hospitalized case)	3 yr.	
27. E. D. (non-hospitalized case) mixed tumor clinically	16 yr.	
28. P. D. (non-hospitalized case)	3 yr.	
29. A. S. (non-hospitalized case)	10 mo.	
30. C. M. (non-hospitalized case)	7 mo.	
31. M. H. (M. H. #41268)	9 mo.	
32. A. L. (J. H. #255333)	.....	Operated upon following trial of x-ray therapy; marked improvement; 5 yr. follow-up
33. I. S. (J. H. #247534)	.....	Operated upon following trial of x-ray therapy; slight improvement; 1½ yr. follow-up; died of cerebral hemorrhage
34. H. S. (N. I. #10515-H) Cystic adenoma	.....	Operated upon; postoperative death

TABLE V

RADIATION THERAPY—FURTHER VISUAL LOSS

Case	Subsequent Treatment	Subsequent Course
35. H. K. (N. I. #16515-H)	Operation; postoperative x-ray therapy	Slight improvement; 13 yr. follow-up
36. L. G. (J. H. #233016)	Operation	Slight improvement; 7 yr. follow-up
37. E. J. (J. H. #265836)	Operation	Marked improvement; 4 yr. follow-up
38. N. G. (J. H. #246192)	Operation	Slight improvement; 2 yr. follow-up
39. B. F. (J. H. #268184)*	Operation; postoperative x-ray therapy	Marked improvement; 4 yr. follow-up
40. A. W. (J. H. #241904) Cystic adenoma	Operation	Marked improvement; 6 yr. follow-up
41. I. W. (M. H. #39760) Mixed tumor	Operation	Slight improvement; 20 mo. follow-up
42. E. G. (J. H. #259616) Cystic adenoma	Operation	Slight improvement; 4½ yr. follow-up
43. C. S. (J. H. #221701) Cystic adenoma	Operation; postoperative x-ray therapy	Marked improvement; 8 yr. follow-up
44. P. C. (J. H. #260198)	Operation; postoperative x-ray therapy	Marked improvement; 3 yr. follow-up
45. P. O. (M. H. #41288)	Operation	Slight improvement; 5 mo. follow-up
46. M. O. (M. H. #39803)	Operation	Marked improvement; 1½ yr. follow-up
47. E. B. (non-hospitalized case)	Refused operation	
48. H. R. (non-hospitalized case (Mixed tumor clinically	Operation elsewhere	
49. C. G. (non-hospitalized case)	Refused operation	Loss of vision one eye; loss of temporal field in other eye with maintenance visual acuity 20/40; 5 yr. follow-up
50. R. F. (J. H. #261704)	Operation	Postoperative clot; vision much worse after operation; unimproved; 4 yr. follow-up
51. A. S. (N. I. #21537)	Operation	Slight improvement postoperatively; died 10 yr. later of recurrence
52. R. M. (J. H. #253448)	Operation	Postoperative death
53. B. A. (N. I. #27341-H)	Operation	Postoperative death
54. S. S. (J. H. #261926)		Prepared for operation; failure of respiration during induction of anesthesia
55. Y. K. (J. H. #237623)	Operation	Marked improvement; recurrence 3 yr. later; no improvement with x-ray therapy; reoperated; postoperative death
56. B. S. (J. H. #270951)† (M. H. 40549)	Operation	Marked improvement; recurrence within 2 yr.; x-ray therapy without improvement; reoperated; improved; beginning regression again within a year following operation

* Return of menses; had baby.

† Return of menses following first operation; had baby.

TABLE V (*Continued*)

Case	Subsequent Treatment	Subsequent Course
57. A. G. (N. I. #12295-H)	Operation	Improved markedly; recurrence 4 yr. later; reoperated; unimproved for 5 yr.; further decline of vision; third operation; postoperative x-ray therapy; amaurosis; death 3 mo. following third operation
58. C. H. (J. H. #283060) Cystic adenoma	Operation	Marked improvement; 2 yr. follow-up

TABLE VI

RADIATION THERAPY—PRE- AND POSTOPERATIVELY—VARYING EFFECTS

Case	Course
59. I. S. (J. H. #264399)	Essentially unimproved following one course x-ray therapy; failure of vision following operation and postoperative x-ray treatment; fourth course of radiation therapy with improvement in one eye and transient improvement in other eye; still another course of radiation therapy with continued improvement in one eye and delayed improvement in other eye

TABLE VII

OPERATION COMBINED WITH POSTOPERATIVE IRRADIATION—RESULTS

Case	Degree of Improvement
60. S. S. (N. I. #12416-H)	Improvement of vision in one eye; other eye remained amaurotic; 4 yr. follow-up
61. M. R. (B. I. #91771)	Marked improvement; 10 yr. follow-up
62. M. K. (N. I. #32889)	Marked improvement; 9 yr. follow-up
63. A. B. (J. H. #222720)	Marked improvement; 8 yr. follow-up
64. H. S. (N. I. #23257-H)	Marked improvement; 4 yr. follow-up
65. J. M. (J. H. #215008) Mixed tumor	Marked improvement; 9 yr. follow-up
66. L. S. (J. H. #246861)	Marked improvement; 6 yr. follow-up
67. M. C. (M. H. #41746)	Marked improvement; 6 mo. follow-up
68. A. T. (J. H. #272125)	Unimproved; 2 yr. follow-up; (severe visual impairment preoperatively)
69. M. T. (N. I. #22488)	Unimproved; 12½ yr. follow-up; (severe visual impairment preoperatively)
70. R. B. (B. I. #84165) Cystic adenoma	Unimproved; 6 mo. follow-up; (severe visual impairment preoperatively)
71. M. K. (J. H. #244685)	Unimproved postoperatively; (severe visual impairment preoperatively); died 10 mo. later as a result of injury
72. L. F. (J. H. #241925)* Mixed tumor	Marked improvement; beginning regression of vision in one eye 6 yr. after operation
73. I. W. (J. H. #208671)	Progressive failure of vision; refused reoperation; received 53,100 r over a 4-yr. period; died 8 yr. postoperatively
74. O. G. (N. I. #14191-H)	Slow progressive visual loss; 15 yr. follow-up
75. T. C. (J. H. #280772)	Slight improvement; recurrence after 6 yr., failure of x-ray therapy to halt visual loss; reoperated upon; death 6 wk. later
76. L. H. (J. H. #222976) Mixed tumor	Slight improvement; recurrence within 4 mo.; reoperation plus x-ray therapy; continued to fail; died 10 mo. later following an operation elsewhere

* Became a father.

sion. One of these patients improved a second time with further radiation therapy. (Case 21, Table III.) Visual failure was halted in an additional 10 patients, 17 per cent, although no improvement in vision occurred. (Table IV.) Nevertheless, since operation was deemed advisable and performed in three of these ten cases, from a practical standpoint, x-ray therapy was efficacious in only seven of them, 12 per cent. Further visual failure despite irradiation occurred in twenty-four cases, 41 per cent. (Table V.) Of twenty-three patients worse or unimproved following radiation therapy and subjected to operation, cystic adenomas were found in only five. The case listed in Table VI was unique in several respects, including the response to roentgen treatment. (Fig. 14.) Preoperative radiation effected little change and following operation vision deteriorated further. Only after a fourth course of irradiation was improvement apparent; in one eye, improvement was of transient duration. A fifth course of radiation therapy was administered; and while improvement in the poor eye did finally occur, it was long delayed. Case 90 included in Table VIII also merits comment. This patient was originally operated upon because a suprasellar meningioma was suspected. What appeared to be a complete removal of the tumor was accomplished and the tissue was reported to be a meningioma. Recurrent symptoms twenty-two months later led to a review of the sections of the tumor at which time the correct diagnosis of a chromophobe adenoma was established. Radiation therapy following symptoms of recurrence resulted in marked improvement which has been maintained for a five-year period.

TABLE VIII

RESULTS FOLLOWING OPERATION ALONE

Case	Course
77. L. B. (J. H. #285402)	Marked improvement; 2 yr. follow-up
78. H. S. (non-hospitalized case) transfrontal operation 1927 by Dr. Cushing	Unimproved; useful vision one eye; 11 yr. follow-up
79. A. B. (non-hospitalized case)* Transfrontal operation 1931 by Dr. Cushing.	Unimproved; 13 yr. follow-up
80. A. W. (M. H. #40077) Transphenoidal operation 1927 by Dr. Cushing.	Maintained vision in right eye; left eye blind; recurrence after 17 yr.; x-ray therapy failed to halt visual loss; operation; postoperative death
81. M. R. (J. H. #279166) Transfrontal operation 1932 by Dr. Cushing Mixed tumor.	Marked improvement; recurrence 12 yr. after operation; irradiation failed to halt visual decline; reoperated; died 1 mo. postoperatively.
82. E. S. (N. I. #33280) Transfrontal operation 1935 by Dr. Frazier.	Recurrence 2 yr. later; reoperated 1937; continued to fail; expired 1941
83. V. D. (M. H. #22174)† Transphenoidal operation 1922 by Dr. Cushing.	Improvement; recurrence in 1927; improvement with x-ray therapy; recurrence in 1930; further irradiation in 1933 without improvement; transfrontal operation with postoperative irradiation in 1933; marked improvement maintained since then
84. I. D. (V. C. #289850) Transphenoidal operation 1911 by Dr. Cushing.	Improved considerably; recurrence in 2 yr.; subtemporal decompression and reoperation through transphenoidal route by Dr. Cushing; improved; recurrence 5 yr. later; almost completely amaurotic in 1931
85. J. Y. (M. H. #41444) Transphenoidal operation 1927 by Dr. Cushing.	Recurrence in 1934; transfrontal operation and postoperative irradiation; improved; recurrence within a yr.; x-ray therapy without improvement; relatively little change until 1946; further regression with mental changes and convulsions; third operation with improvement of mental condition; vision no better
86. J. S. (J. H. #255405)	Postoperative death
87. R. C. (J. H. #210323)	Postoperative death
88. N. A. (N. I. #28059-H) Mixed tumor	Postoperative death
89. A. C. (N. I. #13507-H) Mixed tumor	Postoperative death
90. L. K. (J. H. #217843)	Improved; recurrence after 22 mo.; x-ray therapy; marked improvement; 5 yr. follow-up

* Return of menses; had baby.

† Became a father in 1928.

The results achieved with radiation therapy in this series are shown in Table IX.

In summary then, thirty-five patients (59 + per cent) showed a favorable response to irradiation, varying in degree from an arrest of progressive visual failure to mark improvement of visual acuity and visual fields.

TABLE IX
RESULTS FOLLOWING RADIATION THERAPY

1. Improved	slightly 3	17
	markedly 14	
2. Improvement with subsequent regression		7
3. Unimproved but no worse (Three were subsequently operated upon)		10
4. Varying response on different occasions with eventual improvement (Case 59)		1
5. Further visual failure		24
Total		59

A total of twenty-eight patients were operated upon following radiation therapy. This included some who were unimproved, those in whom vision deteriorated still further and several who manifested evidence of recurrence following a favorable response to radiation. Additional postoperative radiation was administered in four of these patients. The results are listed in Table X.

TABLE X
RESULTS OF OPERATION FOLLOWING RADIATION THERAPY

1. Improved	slightly 8	21
	markedly 13	
2. Progressive failure of vision (Case 59)		1
3. Vision worse following operation (postoperative clot)		1
4. Postoperative death		4
5. Death 4 mo. after operation (pituitary failure)		1
6. Subsequent recurrence		5

There were five patients with recurrence following operation. In four of these, recurrence appeared within four years and it was subsequently necessary to reoperate upon all of them. Concerning the fifth case, detailed information was not available. However, he is known to have expired ten years after operation with evidence of recurrence. Regression in one patient (Case 24) was favorably affected by roentgen treatment for a period of four years. Of the four patients for whom a second operation was performed, one has remained slightly improved; another is showing signs of further recurrence; a third was unimproved and eventually required still another operation which failed to prevent further loss of vision and the patient expired three months later; one died postoperatively.

TABLE XI
RESULTS OF OPERATION COMBINED WITH POSTOPERATIVE RADIATION THERAPY

Improved	slightly 2	10
	markedly 8	
Improved markedly in one eye; remained blind in other eye		1
Unimproved		4
(Vision severely impaired in all 4 cases preoperatively)		
Progressive failure of vision		2
Recurrences		3

In seventeen cases treatment consisted of operation followed by irradiation. (Table VII.) The results obtained are shown in Table XI.

Of the two patients who continued to lose vision, one has done so slowly over a fifteen-year postoperative period. Vision in the second patient deteriorated more rapidly and the patient expired eight years after operation. Reoperation having been refused, an enormous amount of irradiation, 53,100 r over a four-year period, was administered but to no avail. Recurrences occurred in three cases. In one evidence of slight regression occurred recently; and should this be confirmed, in all probability a course of radiation therapy will be recommended. Recurrence in a second case was treated at first with irradiation and when this proved unsuccessful another operation was performed. Death occurred six weeks later. The third patient who suffered from a recurrence was reoperated upon and given roentgen therapy postoperatively but prevention of visual failure was not achieved. He expired following a third operative procedure performed elsewhere.

The cases listed in Table VIII, which records the results following operation alone, do not lend themselves to analysis as a whole. Eight of them had previously been operated upon elsewhere and of these six were seen in this clinic because of recurrent

symptoms. There was considerable variation in their course so that no generalizations can be drawn. Four patients in this group (Cases 77, 86, 87, 90) were operated upon because the diagnosis of pituitary adenoma was not clear. Three showed atypical clinical features which introduced some question as to diagnosis; the fourth was thought to harbor a suprasellar meningioma. Two additional cases, both postoperative fatalities, are included although had they survived, they would undoubtedly have received postoperative radiation therapy.

Exclusive of recurrences, operation was performed in fifty-two cases of the entire series of chromophobe adenomas. There were three postoperative clots all of which were recognized and evacuated. Eight postoperative deaths occurred with a resultant mortality figure of 15.4 per cent. In one of these cases, the tumor could not be visualized at operation by virtue of its location (Case 87, see case report). In another, the tumor had extended into the third ventricle, necessitating a surgical approach through the corpus callosum (Case 86, see case report). Cerebral thrombosis was the cause of death in a third case. A fourth patient was in a semi-comatose state prior to operation. Still another had an extensive intracranial extension along the base of the skull toward the foramen magnum into the posterior fossa. Three patients expired presumably because of postoperative cerebral edema although no permission for postmortem examination was obtained. Reoperation because of recurrence was performed in nine cases. There were two postoperative fatalities. Two patients required a third operation. Both survived the surgical procedure though one expired three months later.

CASE REPORTS

Improvement Following Irradiation. B. L. (Case 2). Jewish Hospital, No. 251856. A thirty-five year old white female was admitted on March 3, 1942, because of headache and progressive loss of vision of four months' duration. She was seven months pregnant. Limitation of the peripheral fields of vision was first noted while driving an automobile.

On examination a bitemporal hemianopsia was demonstrable. Visual acuity on the left was 20/40, on the right questionably 2/200. Roentgen examination of the skull revealed enlargement of the sella turcica with considerable thinning of the posterior clinoids, dorsum sellae and floor. Considering the degree of visual impairment, immediate operation ordinarily would have been the procedure of choice. In view of the fact, however, that the patient had no other children, was thirty-five years old and extremely eager to continue with her already advanced pregnancy, a carefully supervised trial of radiation therapy was instituted. Irradiation was administered in daily doses of 200 r. Three portals, two lateral and one anterior, were treated in rotation, a total of 800 r being given to each field over a period of twelve days. Her visual fields were examined every day. Within a week's time, a distinct improvement in visual acuity and peripheral fields was observed. At the time of discharge from the hospital on March 17, 1942, visual acuity on the left was 20/20 and on the right, 20/100. On April 16, 1942, a second course of radiation therapy was begun and a total of 3,000 r administered. She continued to maintain visual improvement. On May 28, 1942, a normal baby was delivered by cesarean section. Following the birth of her child, a third course of irradiation was prescribed. In June, 1942, visual acuity was 20/20 on the left and 20/40 on the right. The peripheral fields continued to expand. In June, 1943, however, there was some recurrence of headache and a slight shrinkage of the peripheral fields. Another course of roentgen therapy was administered, totalling 3,000 r. Improvement promptly ensued. In September, 1943, menses were resumed. The patient was examined again on March 14, 1946, at which time the right optic disc showed a slight degree of atrophy. Her condition otherwise was very satisfactory. Perimetric studies in July, 1947, revealed full peripheral fields and visual acuity of 20/20 on the left and 20/20 −2 on the right (Fig. 6.)

B. S., (Case 10), non-hospitalized patient. A twenty-seven year old white female was seen on January 29, 1942, because of visual impairment. She had consulted an ophthalmologist in February, 1941, who found a bitemporal hemi-

anopsia and visual acuity of 20/20 on the right and 20/30 on the left. An enlarged sella turcica was also discovered. It was further learned that her menses had begun at the age of eleven but had ceased when she was about fifteen years old. On a few occasions there was some slight staining brought about by the injection of female sex hormone. She had gained about 10 or 12 pounds during the past six to seven years. Radiation therapy was given from April 11 to June 2, 1941, 1,500 r having been administered to each of three portals. The patient stated her eyesight now was worse than at the time treatment was completed.

Examination revealed a short, obese, pale young woman with a thin, dry skin, tapering fingers and fine, silky hair on her head. There was no axillary hair whatever, only a small amount of hair over the labia and practically none over the pubis. The external genitali were

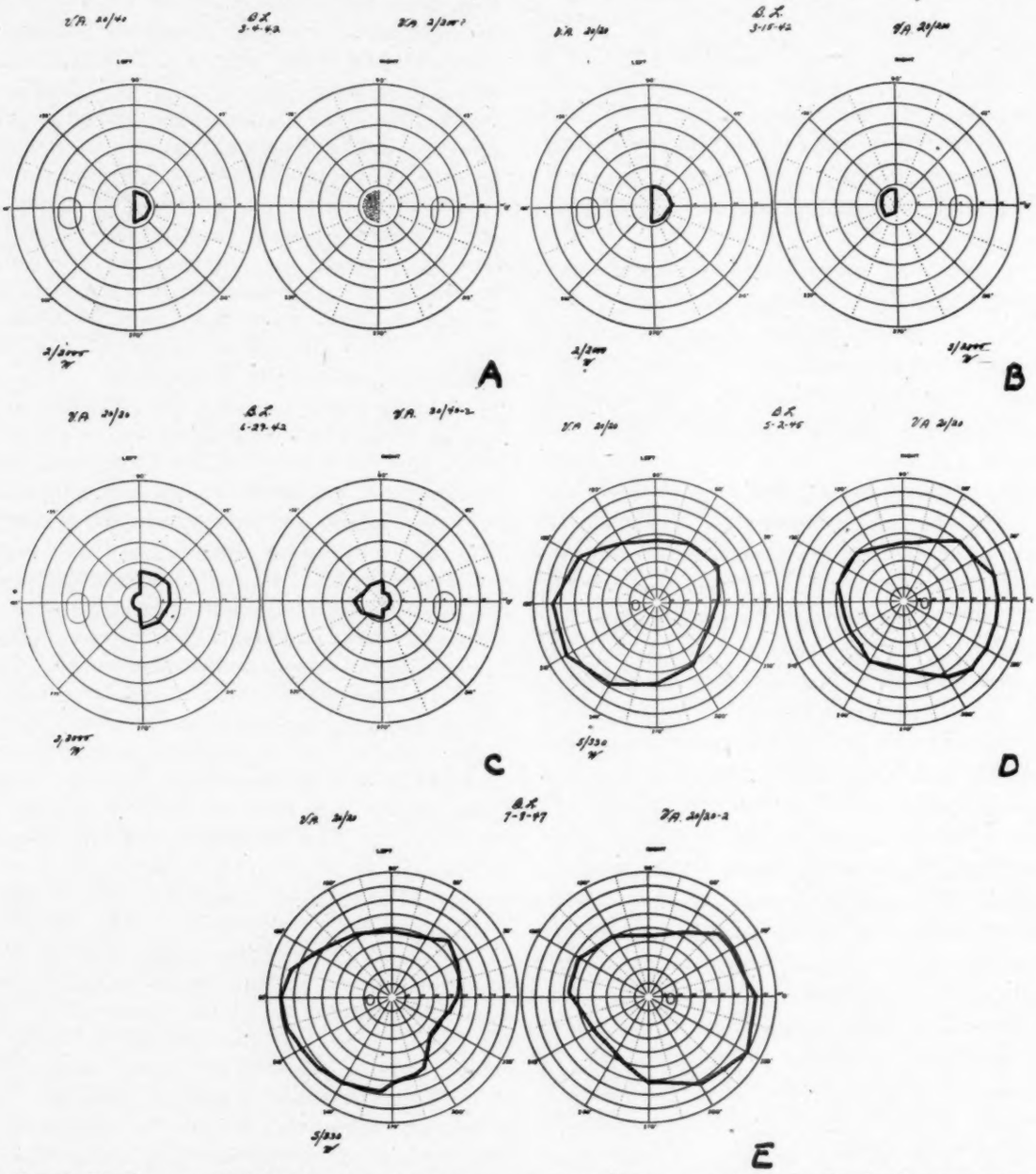


FIG. 6. Case 2. A, central fields prior to irradiation; B, central fields eleven days after instituting treatment; C, central fields following third course of therapy; D, peripheral fields, three years follow-up; E, peripheral fields, five years follow-up.

infantile and the breasts underdeveloped. There was pallor of the optic nerve heads. An x-ray of her skull showed an enlarged sella turcica with a thinning of the posterior clinoids and dorsum sellae. Visual field studies revealed a bitemporal hemianopsia; the visual acuity

A course of radiation therapy was started on February 19, 1946, and completed on April 19, 1946. A total of 3,390 r was applied to three fields. The patient improved subjectively but in August, 1946, further diminution occurred in the visual fields and in the acuity of the left

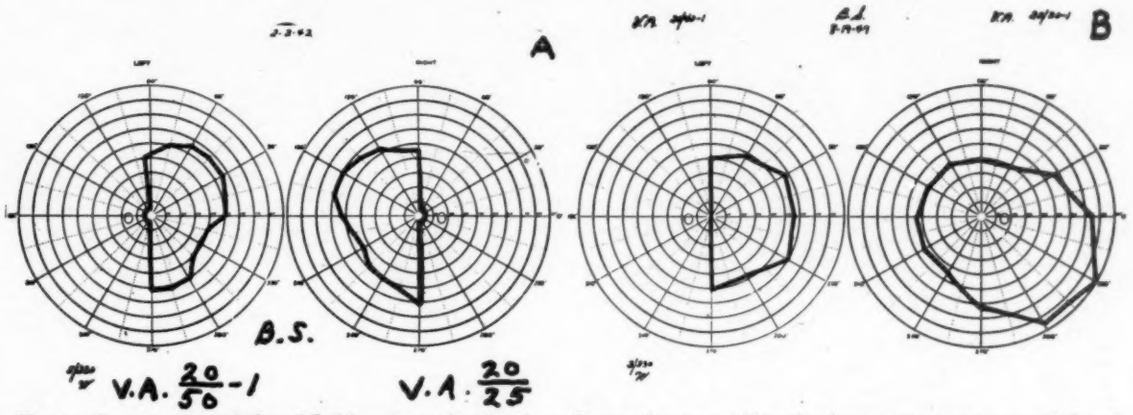


Fig. 7. Case 10. A, peripheral fields prior to institution of second course of irradiation; B, most recent peripheral fields, five and a half year follow-up.

was 20/25 on the right and 20/50 − 1 on the left. A further trial of radiation therapy was deemed advisable and another course administered from February 2 to March 20, 1942. Again 1,500 r were applied to each of three portals. Visual acuity improved and the right temporal field began to expand. There has been little change since 1944. Her visual acuity at present is 20/20 − 1 on the right and 20/40 − 1 on the left. (Fig. 7.) She complains of no headache and in general, feels quite well. The amenorrhea persists.

E. B., (Case 17), non-hospitalized patient. A thirty-nine year old white female was seen on February 18, 1946, because of blurred vision. She was well until three years ago at which time her menses ceased abruptly. She received injections of gonadotropic hormones without any effect on the amenorrhea. A few months previously vision became blurred. She consulted an ophthalmologist who discovered a bitemporal hemianopsia. Headache was never a symptom. There was no disturbance of libido.

Neurologic examination was negative except for the visual field changes. Visual acuity was 15/30 on the right and 15/70 on the left. Funduscopic examination revealed no change in the optic discs. Skull films indicated marked enlargement of the sella turcica with atrophy of the posterior clinoids, sellar floor and dorsum sellae.

eye. She was seen again in this clinic in October and further irradiation recommended. From November 4, 1946, to December 20, 1946, she received a total of 3,300 r. Vision improved gradually over a period of several months. When last seen on September 9, 1947, she was asymptomatic. Visual acuity on the right was 15/20 and on the left, 15/50. The visual fields had improved a good deal. (Fig. 8.)

M. S., (Case 3), Montefiore Hospital, No. 41698. A fifty-two year old white male came under observation in October, 1946, because of visual failure of about one year's duration. Impairment of sight was more pronounced in the right eye. For about a similar length of time, he had experienced intermittent headache, frequently quite severe in intensity. Libido and potency had been diminished for several years, and in the past few months he had been completely impotent. Operation was advised at another hospital but refused by the family.

Examination revealed a pale, somewhat obese patient with a thin skin, sparse beard and tapering fingers. There was slight bilateral optic atrophy. Visual field studies revealed a left homonymous hemianopsia. Visual acuity was 7/200 on the right and 18/200 on the left.

Roentgenograms of the skull showed enlargement of the sella turcica with atrophy of the posterior clinoids, dorsum sellae and sellar floor. Destructive changes were also observed

in the left sphenoid ridge, left fronto-orbital region, left petrous apex and possibly also the right petrous apex. The extensive bony destructive changes raised some question as to the diagnosis, but the composite clinical picture was clearly that of a chromophobe pituitary adenoma.

F. Y., (Case 1), Montefiore Hospital, No. 42294. A forty-nine year old white female was admitted to the hospital on March 19, 1947, because of visual impairment. Eight years ago dimness of vision was first observed. During the year prior to admission, it became increasingly difficult for her to read. She complained of no

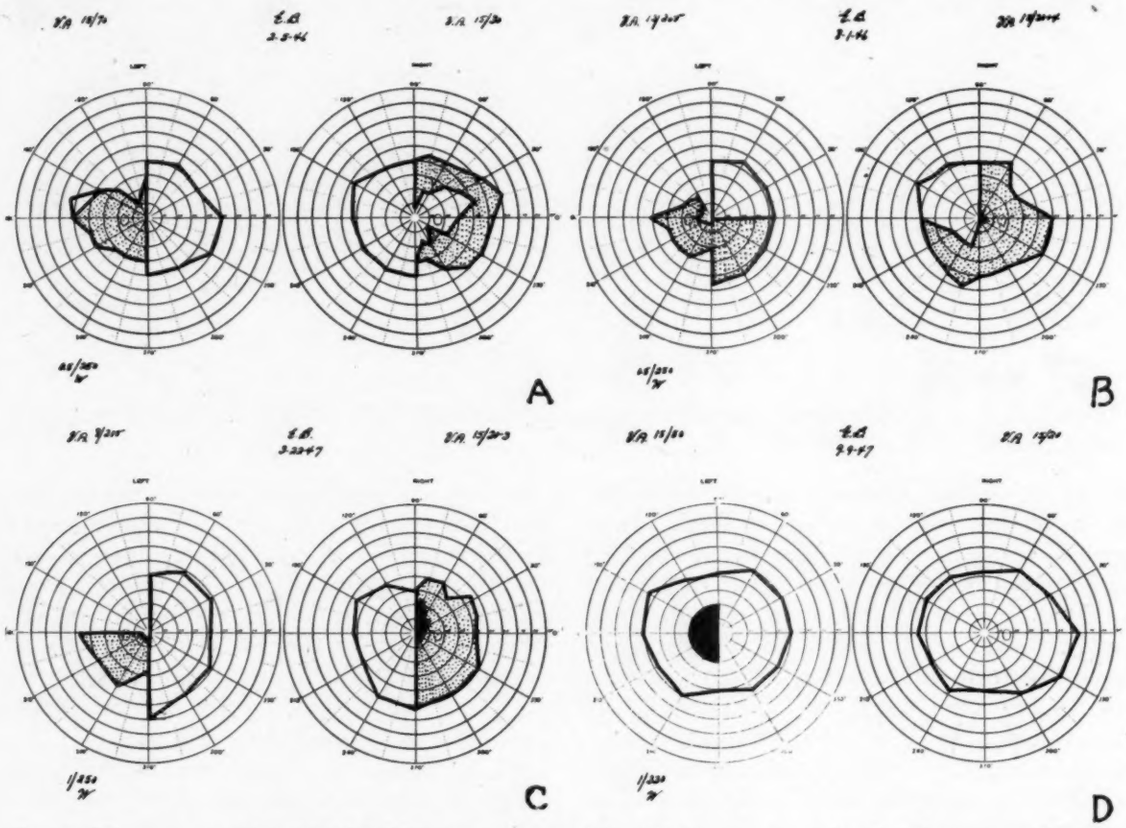


Fig. 8. Case 17. A, peripheral fields before therapy; B, peripheral fields, three months after first course of irradiation; C, peripheral fields, three months after second course of therapy; D, most recent fields, six months later.

Radiation therapy was instituted and in October and November, 1946, he received 2,925 r. There was some improvement in visual acuity on the left but headache was worse. Surgical intervention was again proposed and the patient scheduled for operation. During the induction of anesthesia, however, his condition became precarious and the procedure was cancelled. Further irradiation was, therefore, prescribed and two subsequent courses, each of 3,000 r, were administered between December 27, 1946, and January 31, 1947, and between March 10, 1947, and April 11, 1947. Visual acuity and fields showed remarkable improvement. On September 18, 1947, his vision was 15/20 in both eyes and the peripheral fields had expanded considerably. (Fig. 9.) He was completely relieved of headache.

headache. Menses were regular though there had been a period of amenorrhea between the ages of thirty-eight and forty-two.

Neurologic examination revealed pallor of both optic discs and a bitemporal hemianopsia. Visual acuity in the right eye measured 15/100 − 1 and 15/200 in the left eye. Roentgenograms of the skull showed considerable enlargement and atrophy of the sella turcica.

Radiation therapy was instituted and between March 25, 1947, and April 29, 1947, a total dose of 2,900 r was administered. There was slight but definite improvement in vision. On April 3, 1947, visual acuity in the right eye was 15/70 + 2 and the visual fields had expanded slightly. On August 26, 1947, visual acuity on the right measured 20/40 and on the left 20/100. There was further improvement in

the peripheral fields. (Fig. 10.) A second course of irradiation has been started.

Further Visual Failure Following Irradiation Therapy. N. G., (Case 38), Jewish Hospital, No. 246192. A twenty-eight year old white male was first admitted to the hospital on August 21, 1941, because of headache of a little over one year's duration. For two months prior to admission the severity of the headache had increased markedly and was accompanied by visual impairment. He particularly noted that while playing ball he was unable to see the ball from various positions. There was also dimness of vision in the left eye.

On examination, pallor of both optic discs was noted. Visual field studies revealed a bitemporal hemianopsia, more marked on the left. Visual acuity was 20/30 − 2 on the right and 20/70 − 2 on the left. Skull roentgenograms showed a marked ballooning of the sella turcica with loss of density of the sellar floor and posterior displacement of the dorsum sellae. Radiation therapy was begun on August 26, 1941, and while this treatment was being given, visual field studies were continued. He was discharged from the hospital on August 28, 1941.

Visual failure was progressive despite irradiation and after 4,500 r had been administered he was readmitted on October 5, 1941. Visual acuity was 20/30 + 2 on the right and 5/200 on the left. A right transfrontal craniotomy was performed on October 6, 1941, and an intracapsular partial removal of a pituitary tumor accomplished. Microscopic examination of the tumor revealed it to be a chromophobe adenoma. Postoperatively he received a course of irradiation totalling 3,000 r which was completed in November, 1941. Headache was relieved and he felt much improved. Libido and potency were diminished but present. The right central field of vision improved considerably and visual acuity improved to 20/20. There was no change of visual acuity on the left and no restitution of the temporal field on that side. When last seen in October, 1943, his condition and visual status were the same. (Fig. 11.)

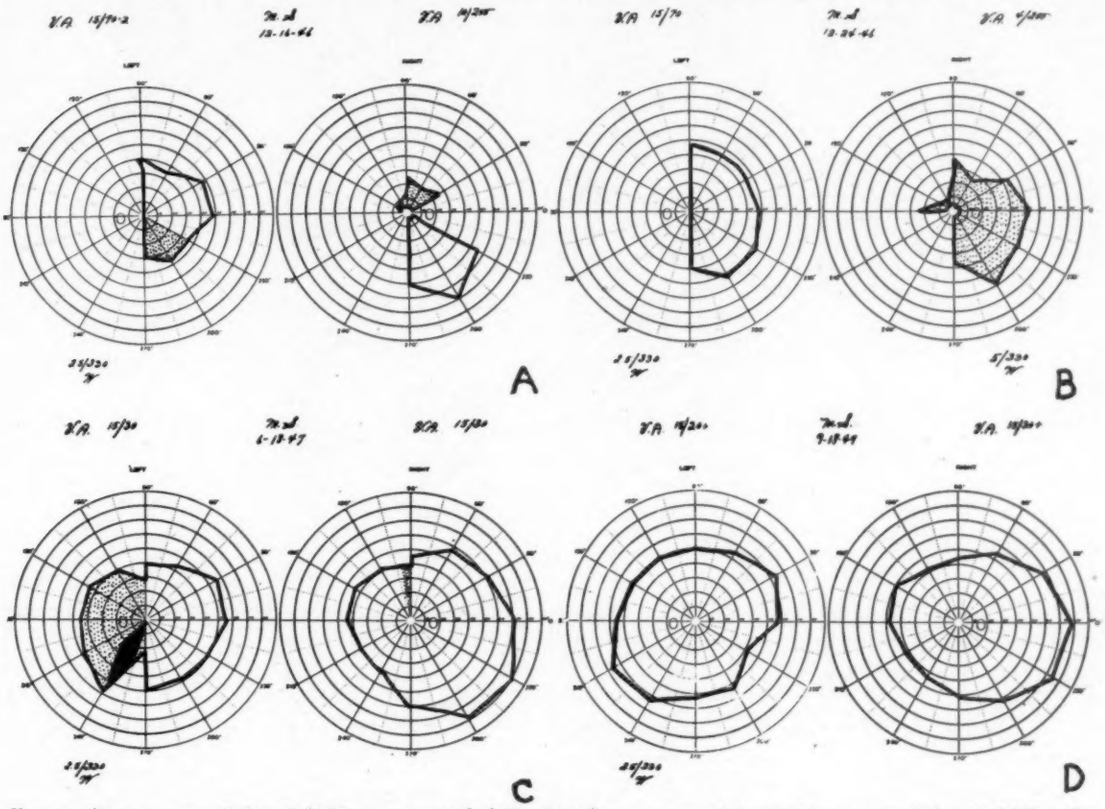


Fig. 9. Case 3. A, peripheral fields, one month following first course of irradiation; B, peripheral fields prior to second course of treatment; C, same, two months after third course of therapy; D, most recent peripheral fields, five months after completion of irradiation.

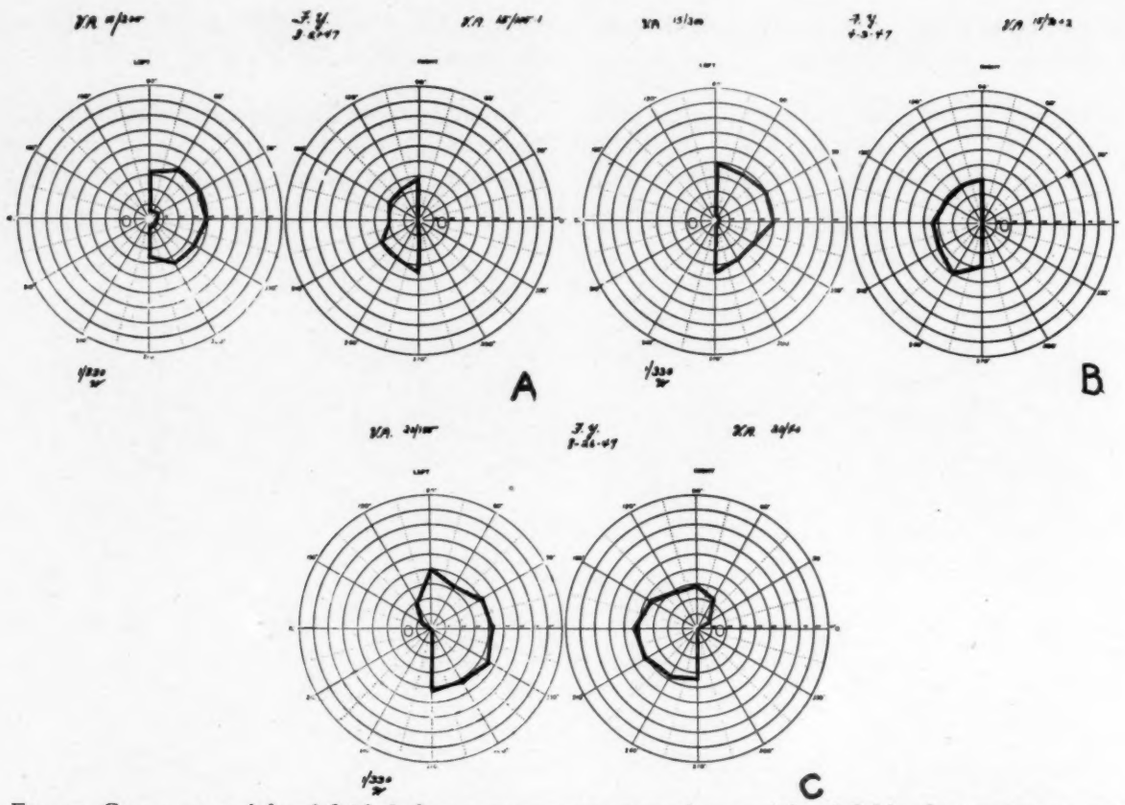


FIG. 10. Case 1. A, peripheral fields before treatment was started; B, peripheral fields after treatment was begun; C, peripheral fields, four months, after completion of therapy.

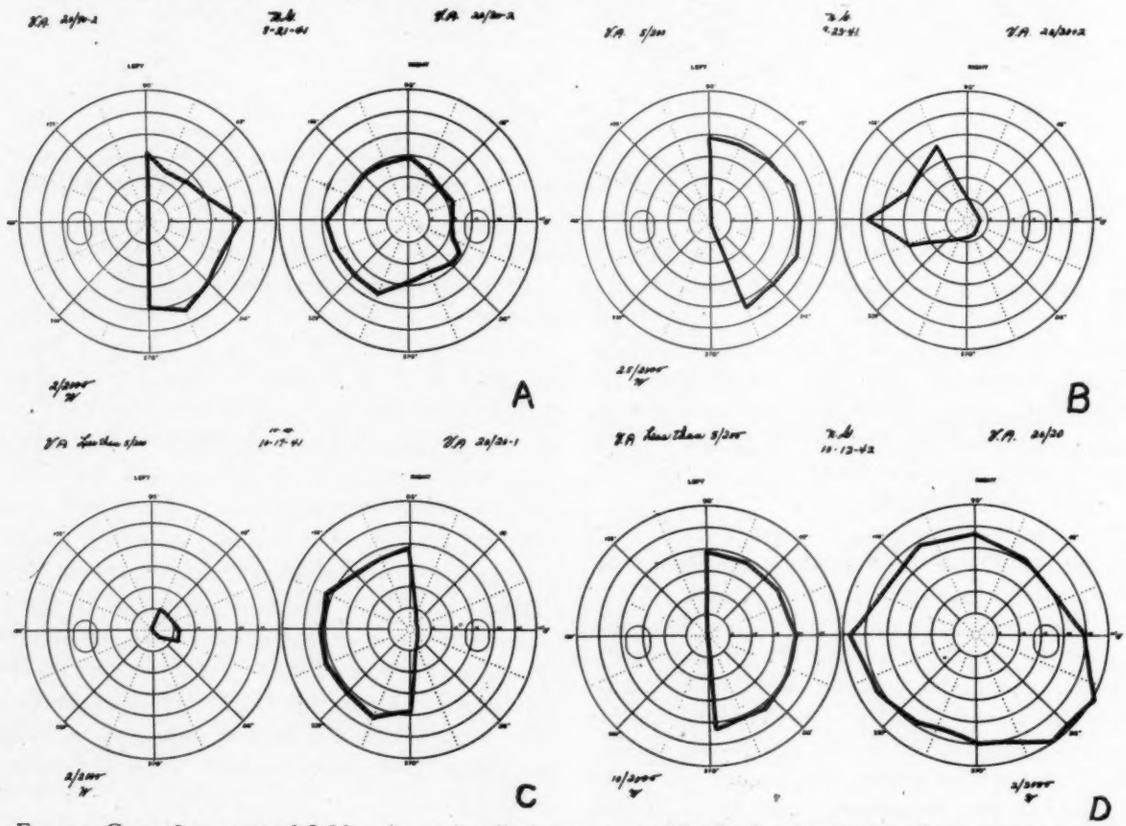


FIG. 11. Case 38. A, central fields prior to irradiation; B, central fields after institution of treatment; C, same after operation; D, central fields one year after operation and postoperative irradiation.

Improvement Following Operation and Postoperative Irradiation. M. C., (Case 67), Montefiore Hospital, No. 41746. A thirty-eight year old white male was admitted on January 28, 1947, because of blurring of vision in the left eye of about one year's duration. This was his only complaint. He denied any change in his body configuration or alteration in libido or potency. He always had a rather sparse beard and a limited amount of body hair. He was married and had three children.

Examination revealed a well developed but pale man with a completely negative neurologic status except for the visual field studies. These revealed a bitemporal hemianopsia, more advanced in the left eye. Visual acuity was 15/15 in the right eye and 15/40 − 2 in the left eye. Roentgenograms of the skull showed a thick-

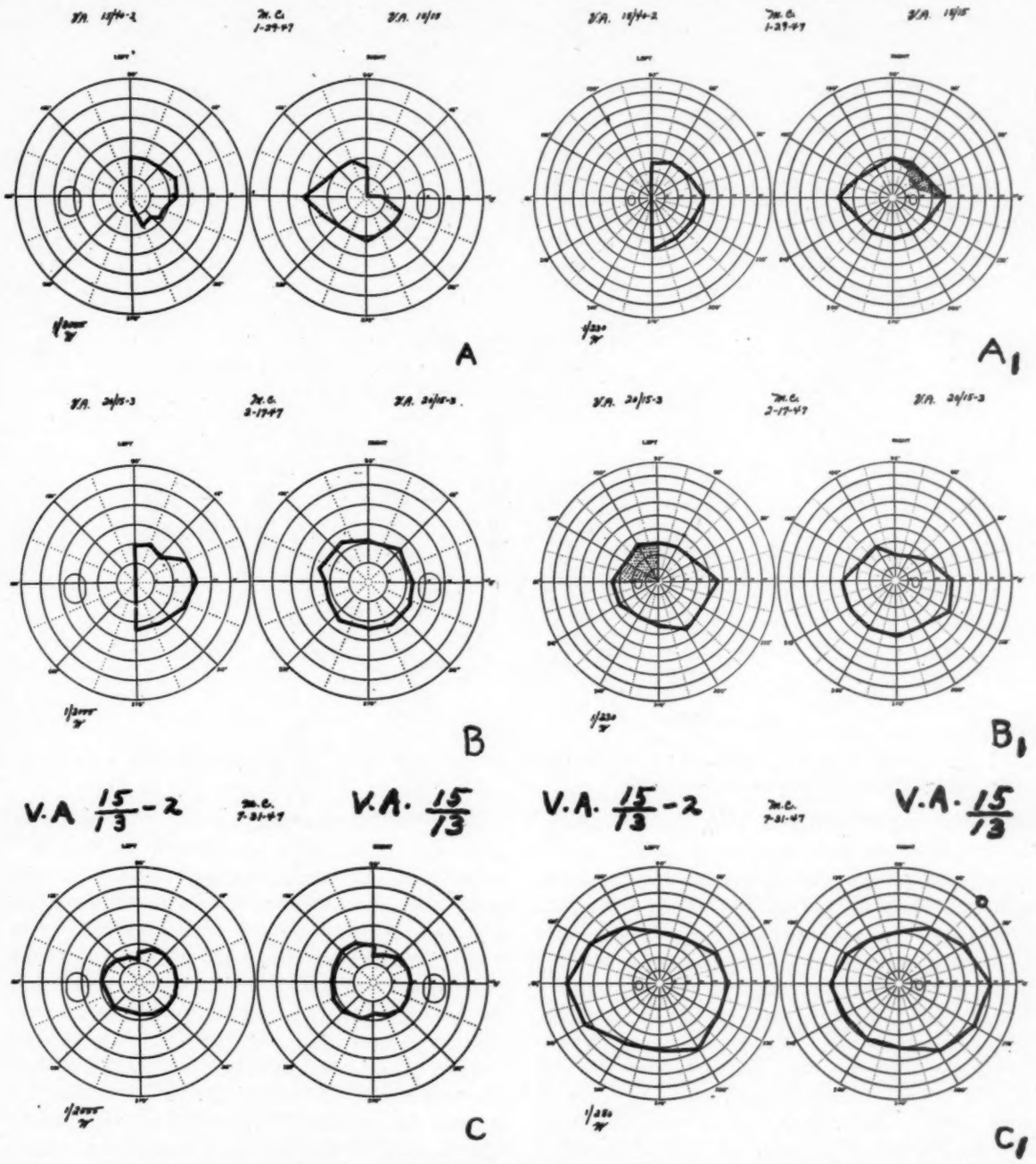


FIG. 12. Case 67. A-A1, central and peripheral fields prior to operation; B-B1, postoperative central and peripheral fields; C-C1, same four, months after completion of irradiation.

ened vault, and *a sella turcica of normal size*, but with atrophy of the posterior clinoids and dorsum sellae. Pneumencephalography was performed revealing a chiasmal mass continuous with the sellar contents, obliterating the cisterna chiasmatis and the anterior half of the cisterna interpeduncularis. (Fig. 2.) In view of the fact that the sella was not enlarged, operation for purposes of verifying the nature of the lesion appeared indicated. On February 3, 1947, a right transfrontal craniotomy was performed, a chiasmal tumor encountered and a radical intracapsular removal accomplished. Examination of the tissue revealed it to be a chromophobe adenoma. Postoperative radiation therapy was given from February 26 to March 18, 1947, the amount being 1,000 r to each of three portals. Since then, visual acuity, central and peripheral fields have shown progressive improvement. Visual acuity at present is 15/13 on the right and 15/13 − 2 on the left. Central fields show slight remains of a defect in both upper temporal regions. (Fig. 12.) The patient is asymptomatic.

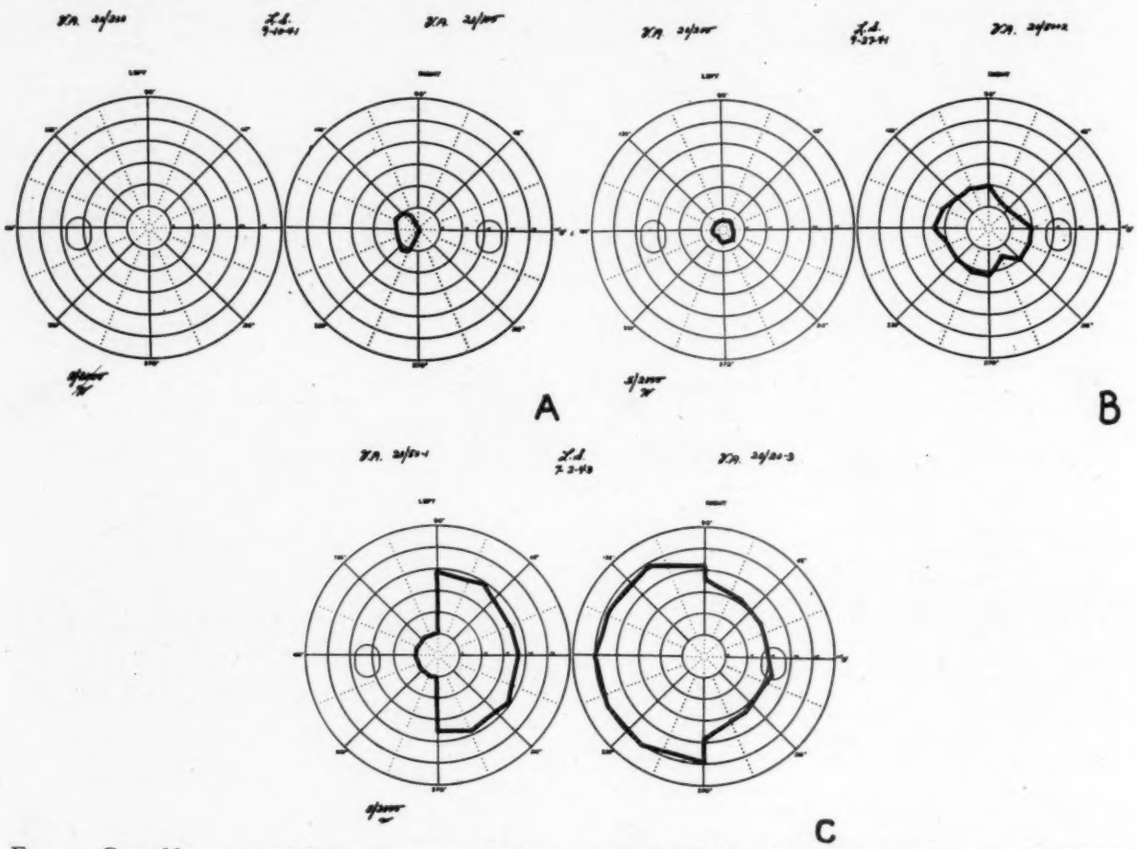


FIG. 13. Case 66. A, central fields prior to operation; B, central fields following operation; C, same, two years after operation and postoperative irradiation.

L. S., (Case 66), Jewish Hospital, No. 246861. A fifty-four year old female was admitted on September 10, 1941, because of headache of four years' duration and blurring of vision of four week's duration. Impairment of sight had become rapidly worse.

Examination revealed an obese woman with a smooth skin and a paucity of axillary and pubic hair. Visual acuity was 20/100 on the right and 20/200 on the left. A bitemporal hemianopsia was demonstrable grossly. Her basal metabolism was −27 per cent. Roentgenograms of the skull revealed enlargement of the sella turcica with destruction of the dorsum sellae and posterior clinoids. In view of the marked visual loss, operation for the purpose of decompressing the optic chiasm was deemed urgent. Accordingly on September 12, 1941, a right transfrontal craniotomy with intracapsular partial removal of a pituitary tumor was performed. The histologic diagnosis of the tumor was a chromophobe adenoma. At the time of discharge from the hospital on September 29, 1941, visual acuity and central fields

had improved in the right eye. In October, 1941, there was beginning improvement in the left eye also. Two courses of radiation therapy, each totalling 3,000 r, were administered postoperatively in October and December, 1941. In June, 1942, another course of irradiation was given, the dose again being 3,000 r. Visual acuity in the right eye improved to 20/20 − 3 and in the left eye to 20/50 − 1. (Fig. 13.) The patient was last seen in July, 1947, at which time her vision was essentially unchanged, and except for an occasional headache, she was feeling well.

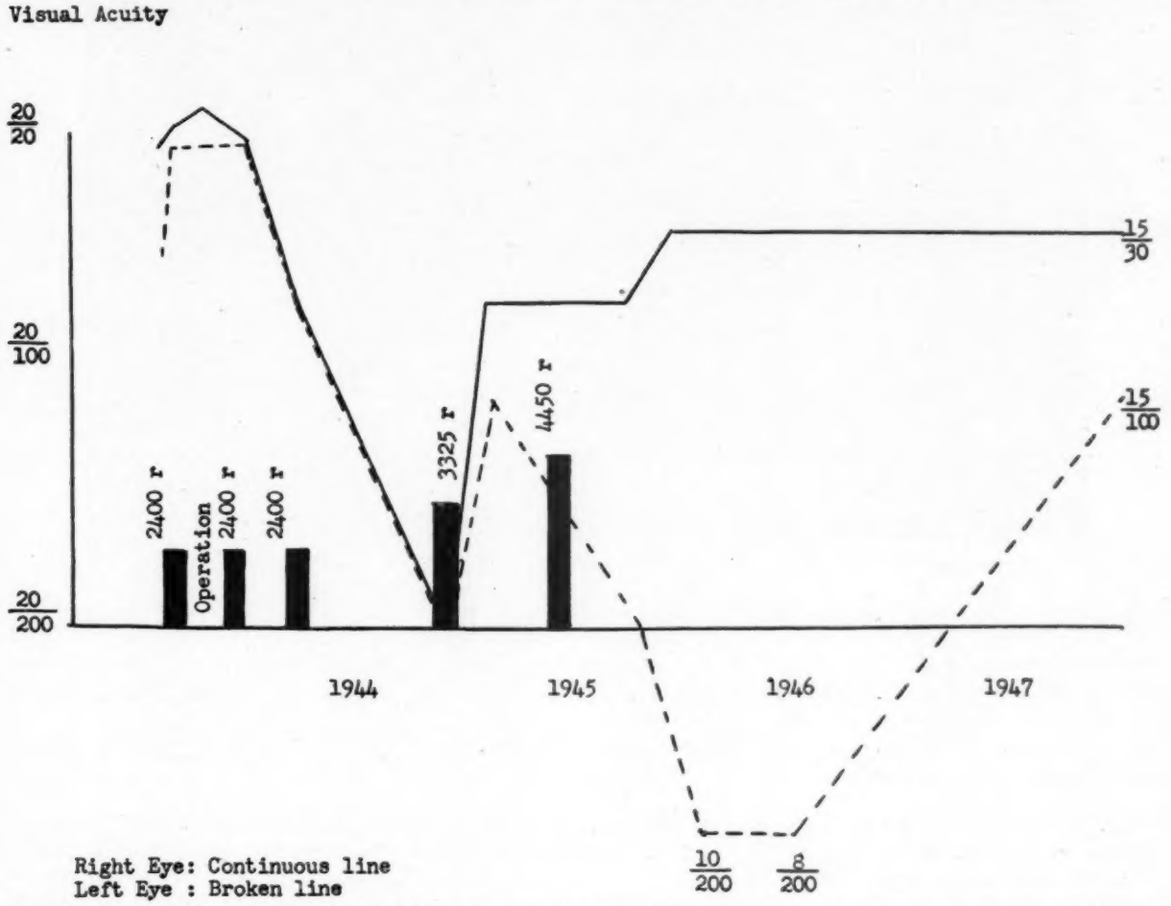


Fig. 14. Case 59. Graph showing the fluctuation of visual activity in each eye in relation to roentgenologic and surgical treatment.

Radiation Therapy Pre- and Postoperatively with Varying Effects. I. S., (Case 59), Jewish Hospital, No. 264399. A fifty-two year old white male was admitted on May 2, 1943, because of diminishing vision of three years' duration. He had been examined in 1940 by an ophthalmologist who found a visual acuity of 20/20 in the right eye and 20/30 + 4 in the left eye. Visual fields showed a suggestive upper temporal slant for color in the right eye. Funduscopic examination revealed no pathologic changes. Skull roentgenograms were reported to have shown enlargement of the sella turcica with thinning of the sellar floor and erosion of the dorsum sellae and posterior clinoid processes. A pituitary neoplasm was suspected. In 1942, the patient began to experience severe headache, confined mainly to the right side. Visual acuity had become more severely affected since January, 1943.

Examination revealed a tall, thin man without any evidence of hypopituitarism. Bilateral optic atrophy of moderate degree was noted. Visual acuity on the right was 20/30 − 1 and on the left 20/50 − 1. Visual field examination revealed an unusual picture. There was an inferior altitudinal defect on the right and an inferior nasal slant on the left side. Roentgenograms confirmed the findings previously noted.

During May, 1943, a course of radiation therapy was given and 2,400 r applied. Visual acuity improved from 20/30 − 1 to 20/20 in the right eye, and from 20/50 − 1 to 20/30 + 2 in the left eye. The visual fields remained unaltered. It seemed most probable that the underlying lesion was a pituitary adenoma, but in view of the incongruous visual fields operation was advised. On June 17, 1943, a right transfrontal craniotomy was performed and a pituitary adenoma disclosed. An extension of the tumor was observed to compress and displace the right optic nerve upward, thus, in all probability, accounting for the inferior visual field defect. Histologic examination of the tissue confirmed the diagnosis of a chromophobe adenoma.

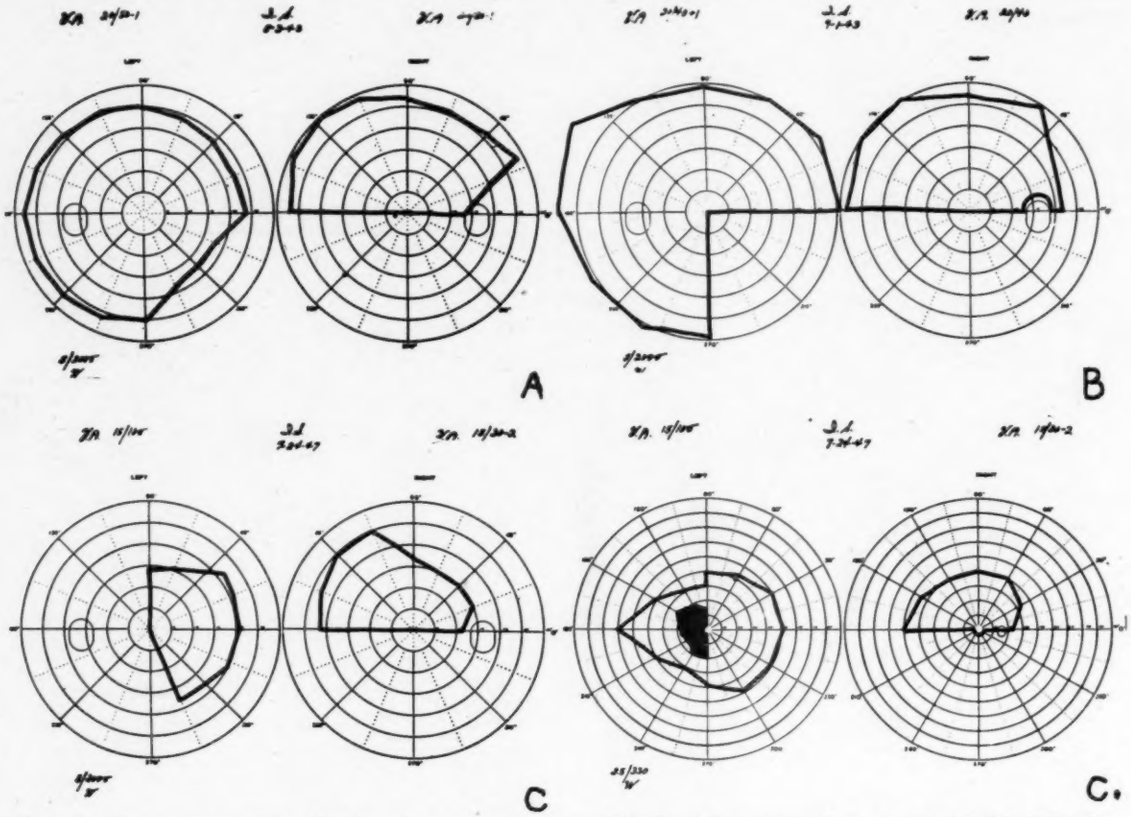


FIG. 15. Case 59. A, central fields prior to treatment; altitudinal hemianopsia; B, central fields following operation and postoperative irradiation; C-C1, most recent central and peripheral fields. Persistence of inferior hemianopsic defect.

Postoperative radiation therapy was instituted and a course of 2,400 r completed on July 15, 1943. Visual acuity, however, proceeded to decline. It was 20/40 in both eyes on September 1, 1943. In October, 1943, another course of radiation therapy was instituted and 2,400 r applied. On December 29, 1943, visual acuity was 20/70 bilaterally. Between May 31, 1944, and July 3, 1944, a fourth course of roentgen treatment was prescribed and 3,325 r administered. In June, 1944, visual acuity was 20/200 in both eyes. Very shortly thereafter, visual acuity began to improve and in September, 1944, measured 20/70 in the right eye and 15/100 in the left eye. Subsequently, however, regression of acuity of vision in the left eye occurred. Between November 27, 1944, and January 12, 1945, a fifth course of irradiation was given, and 4,450 r applied. Further improvement occurred in the right eye but the deterioration of vision in the left eye continued. It reached a low level of 8/200 in February, 1946, and thereafter began to improve.

When examined on July 24, 1947, visual acuity was 15/30 − 2 in the right eye and 15/100 in the left eye. (Figs. 14 and 15.) Hypopituitary stigmas were evident. The patient was pale, his hair very thin and libido was absent. He had been receiving 25 mg. of testosterone once a week for a period of two years. The only effect of this medication was to

Fig. 16. Case 87. Sagittal section showing the tumor behind the optic chiasm projecting up into the third ventricle.

lessen his feeling of fatigue. He complained of no headache and was working at his occupation of a civil engineer.

Atypical Cases. R. C. (Case 87), Jewish Hospital, No. 210323. A fifty year old white male was admitted on March 21, 1938, because of impairment of memory and generalized weakness of one and a half year's duration. He had also begun to manifest emotional changes, consisting of profound depression and episodes of weeping.

Neurologic examination revealed a slight tremor of the extended fingers, some slowness of speech and possibly pallor of the temporal halves of the optic discs. Visual field studies were not altogether satisfactory but were suggestive of a bitemporal hemianopsic defect. Visual acuity measured 20/40 in the right eye and 20/30 in the left eye. Roentgenograms of the skull were negative; the sella turcica was not enlarged or atrophic.

The preponderance of mental symptomatology suggested a presenile type of degenerative disease. However, it was learned that the patient had been hospitalized elsewhere and a pneumencephalogram performed. The films showed a deformity of the cisterna interpeduncularis by a mass which also projected into the third ventricle. (Fig. 3.) A suprasellar tumor, possibly a meningioma, was considered.

On March 24, 1938, a right transfrontal craniotomy was performed and the region of the chiasm explored. Both optic nerves and the diaphragm of the sella turcica were well visualized but no tumor was seen.

The patient failed to recover postoperatively and expired the same day. Postmortem examination revealed a large tumor between the legs of the cerebral penduncles, extending upward and deforming the floor of the third ventricle. (Fig. 16.) The mass was behind the optic chiasm in a location which rendered it surgically unapproachable. Though entirely intracranial, the tumor was of pituitary origin since histologically it revealed the structure of a chromophobe adenoma.

J. S., (Case 86), Jewish Hospital, No. 255405. A fifty-seven year old white male was admitted on June 29, 1942, because of headache, impairment of gait, urinary incontinence and drowsiness. About five years previously, he had first experienced drowsiness but was nevertheless able to continue to work up until fifteen months before. At that time the drowsiness became more marked and, in addition, he became

unable to walk and developed headache. During the past three months, he was incontinent of urine.

Examination revealed a well nourished, drowsy, apathetic patient, responding only after much prompting. Orientation and memory were defective. Objects were named correctly. The right pupil was a trifle larger than the left; both reacted sluggishly to light. No extraocular muscle weakness or conjugate gaze impairment was detected. There was a right central facial weakness with hyper-reflexia and a positive Babinski toe sign on the right. Sensory examination was unreliable. The fundi showed hazy disc margins. Visual field studies and determination of visual acuity were not feasible.

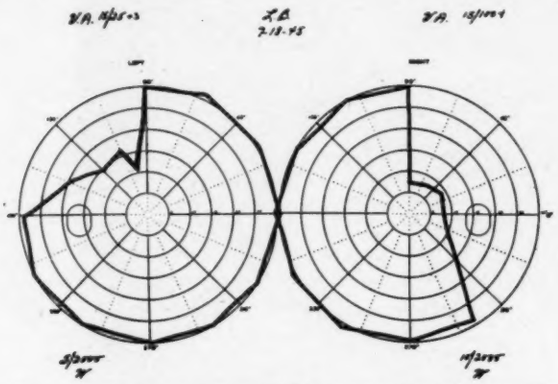


FIG. 17. Case 77. Central fields showing bitemporal hemianopsia, more pronounced on the right.

A ventriculogram was performed on June 30, 1942. This revealed considerable dilatation of the lateral ventricles and a deformity of the anterior portion of the third ventricle outlining a tumor mass. Concave filling defects in the floors of the frontal horns and an elevation of the cisterna interpeduncularis were also demonstrable. (Fig. 5.) The films showed, in addition, marked enlargement of the sella turcica with destruction of its bony boundaries. These findings were interpreted as being indicative of a tumor reaching into the third ventricle, possibly originating from the pituitary. A right transfrontal craniotomy extending across the midline was performed. Between the legs of the optic chiasm a mass was exposed and partially removed. It was evident that insufficient tumor could be removed through this approach. The mesial aspect of the hemisphere was, therefore, retracted, and after incising the corpus callosum, the lateral ventricle was entered. Tumor tissue was seen projecting through the foramen of Monro. A considerable amount was removed, relieving the plug in the foramen and exposing the floor of the third ventricle. Histologic examination of the tissue revealed it to be a chromophobe adenoma.

The patient failed to recover from the operation and expired on the following day.

L. B., (Case 77), Jewish Hospital, No. 285402. A forty-three year old white female was admitted on July 16, 1945, because of headache, diplopia and impairment of vision of the right eye. Her illness began abruptly on May 18, 1945, with intense headache, drooping of the right lid and double vision. She was admitted to another hospital on May 21, 1945, where examination revealed ptosis of the right lid, paralysis of upward and inward movement and weakness of outward movement of the right eye. There was questionable diminution of pinprick appreciation over the ophthalmic division of the right trigeminal nerve. Mentally she was lethargic. Blood pressure was 165/85 mm. mercury. Red blood count was normal; the white blood count was 16,800, with a preponderance of polymorphonuclear leukocytes. A lumbar puncture done on May 21st revealed uniformly bloody cerebrospinal fluid containing over 4,000 red blood cells per cu. mm. The protein content of the cerebrospinal fluid was 90 mg./100 cc. The blood and cerebrospinal fluid Wassermann were both negative. Roentgenograms of the skull showed an enlargement of the sella turcica with thinning of the posterior clinoid processes. Visual fields revealed a constriction of the right temporal field. Electroencephalographic studies were essentially normal. While still in the hospital, the ocular palsies began to improve, but concomitantly progressive bilateral papilledema with hemorrhages appeared. The diminution of sensation over the first division of the right trigeminal nerve became more definite. Mentally, however, she was more alert. On June 22, 1945, the abnormal neurologic findings consisted of bilateral papilledema, a right partial ptosis, a smaller right pupil, weakness of elevation of the right eye, a right temporal field defect and diminished sensation over the right opthalmic distribution.

In view of the sudden onset of an ophthalmoplegia together with subarachnoid bleeding, the question of an aneurysm was given consideration. The combination of progressive papilledema together with disappearance of the

focal ocular palsies, however, was thought to be unusual for an aneurysm.

When admitted to this clinic on July 16, 1945, her neurologic status was unchanged from that previously noted except for the additional finding of weakness of abduction of the right eye. Visual field studies indicated a bitemporal hemianopsia, more marked on the right. (Fig. 17.) The visual acuity was 15/100 −1 in the right eye and 15/25 +3 in the left eye. The impression gained from the plain films of the skull was that the sella turcica was not enlarged but that there was marked atrophy of the dorsum sellae, sellar floor and posterior clinoid processes and that these changes were due to increased intracranial pressure. A ventriculographic study was carried out and this revealed slight dilatation of both lateral ventricles, without displacement, elevation of the anterior aspect of the third ventricle, and a tumor arising from the chiasmal region protruding into the cisterna interpeduncularis. On laminagraphic section, the tumor was seen to erode the anterior wall and floor of the sella turcica and to fill the sphenoidal sinus; enlargement of the sella turcica was clearly apparent. (Fig. 4.)

On July 19, 1945, a right transfrontal craniotomy was performed and a mass was seen presenting itself between the optic nerves. A subtotal removal was accomplished with adequate decompression of the optic chiasm. Histologic study of the tumor revealed it to be a chromophobe adenoma.

The patient improved considerably postoperatively and the papilledema slowly disappeared. In September, 1946, her neurologic status was negative. She complained of occasional headache and diplopia when very tired. Visual field studies on September 18, 1947, still showed bitemporal defects. The visual acuity measured 15/20 +2 in both eyes.

CHROMOPHILE ADENOMAS

In contrast to the number of chromophobe adenomas that came under observation, the cases with acromegaly were much fewer. There were thirty-five eosinophilic tumors so that the proportion of chromophobe to chromophile neoplasms in this series is about 3:1. The sex distribution was not especially unusual, twenty cases having occurred in females and fifteen in men. In general, the patients with acromegaly sought medical advice at an earlier age than did those with chromophobe tumors. Twenty-four of the thirty-five patients were in the fourth decade of life or younger when first seen. Of the remainder, nine were in the fifth decade. Although it was frequently difficult to establish with certainty the date of onset of either illness, the impression gained was that patients with eosinophilic adenomas manifested initial symptoms at an earlier age than did those with chromophobe tumors. Overgrowth of acral parts, which characterizes the disease, was present to varying degrees in all cases. A history was elicited of enlargement of the hands and feet made apparent by the necessity of procuring gloves and shoes of increasingly larger size, of growth and greater prominence of the lower jaw, together with separation of the teeth, of increase in size of the nose, lips, brows and tongue, resulting in the characteristic coarse facial features, and of thickening and swelling of the fingers.

In addition to symptoms of acral enlargement, a very common complaint was headache. It was present in twenty-two cases and often constituted the most distressing feature of the patient's illness. Amenorrhea or oligomenorrhea occurred in fifteen of the twenty female patients. In a few instances, normal menstrual periods were established following treatment. This took place in one patient following radiation therapy; in another, four years after operation and postoperative irradiation. The latter patient subsequently bore a child. In a third case, menses occurring regularly at intervals of two months were established following operation. Menstrual periods were resumed in still another patient after treatment with female sex hormone. Among the fifteen male patients, there were nine with diminution of libido, potency or both. One patient, whose libido was practically nil, reported striking improvement following the use of methyl testosterone. Potency was improved in another case following radiation therapy to

the pituitary. In comparison with the group of patients with chromophobe adenomas, the incidence of symptomatic visual impairment among the acromegalics was very low. It occurred in only six patients, one of whom was found to have papilledema and vascular retinal changes in association with hypertension. Two patients complained of transient diplopia but in neither did the examination reveal an extraocular muscle weakness. A bitemporal hemianoptic defect was demonstrable in one, and a very questionable similar defect in the other. Overgrowth of hair and excessive perspiration were mentioned in the protocols of about one-third of the cases although the actual frequency of their occurrence may have been greater. A variety of other symptoms was elicited. These included generalized weakness, easy fatiguability, joint pains, backache, gain or occasionally loss of weight, polydipsia and polyuria (in association with diabetes mellitus), somnolence, insatiable appetite, propensity for sweets, deepening of the voice and paresthesias.

The characteristic bodily changes, resulting from acral overgrowth and diagnostic of the disease, have already been mentioned. Studies of the visual fields indicated defects in seven cases. A bitemporal hemianopsia was demonstrated in six patients and a homonymous hemianopsia in one. Optic atrophy was observed in four of the cases. The patient with papilledema had ventriculography performed with negative findings, thus excluding an extrasellar tumor. The funduscopic changes were, therefore, interpreted as indications of a hypertensive neuroretinopathy.

Roentgen examination of the skull in these acromegalics revealed thickening of the calvarium, overdevelopment of the paranasal sinuses, prognathism, and usually enlargement of the sella turcica with thinning and depression of the floor, and erosion and posterior displacement of the posterior clinoid processes and dorsum sellae. In five cases, however, the size of the sella turcica was within normal limits. When roentgenograms of the extremities were available, tufting of the terminal phalanges of the fingers was often noted. An increased basal metabolic rate averaging +24 per cent was found in half the cases in which such an examination had been performed. There were a few instances, on the other hand, in which it was abnormally low. There was evidence of disturbed carbohydrate metabolism, either an elevated fasting blood sugar level or an abnormally high glucose tolerance curve in six patients. Hypertension was a complicating factor in four of the thirty-five patients.

Treatment. The same plan of treatment was adopted for the eosinophilic adenomas as was employed in the chromophobe cases. Whenever therapy was indicated, irradiation was prescribed; and only if it failed to achieve its purpose, was operation undertaken. The results obtained in the individual cases are recorded in Table XII.

Twenty-nine patients were treated initially with irradiation and of these, improvement, measured in terms of symptomatic relief and cessation of acral growth, was achieved in eighteen. There was questionable progression of the acromegaly in three patients although one who had complained of headache was relieved of this symptom. Eight patients were unimproved and operation performed in six. Improvement following radiation therapy was obtained in a patient with recurrent symptoms who had been operated upon twice previously. Thus, of a total of thirty patients treated with irradiation, unequivocally favorable results were achieved in nineteen or 63 per cent. Operation following unsuccessful radiation therapy resulted in a cessation of visual failure in one case and in improvement in two other cases, without visual impairment, one of whom received postoperative roentgen treatment. Headache persisted in two patients after operation. In one the headache was probably of functional origin. It is too soon after treatment to evaluate the results in another patient.

TABLE XII

CHROMOPHILE ADENOMAS—TREATMENT AND RESULTS

Case	Therapy	Course
1. I. T. (non-hospitalized case)	Radiation therapy	Normal vision; relief of headache; ? progression of acromegaly; 1½ yr. follow-up
2. R. B. (non-hospitalized case)	Radiation therapy	Very slight progression of acromegaly over a 20 yr. period; normal vision
3. B. G. (non-hospitalized case)	Radiation therapy	Relief of headache and drowsiness; vision normal; cessation of acral growth prior to therapy; 3 yr. follow-up
4. H. R. (J. H. #276441)	No indication for therapy	Jacksonian seizures; negative pneumencephalogram
5. H. B. (non-hospitalized case)	Radiation therapy	Relief of headache; no progression of acromegaly; vision preserved; 3 yr. follow-up
6. R. C. (non-hospitalized case)	Radiation therapy	Relief of headache; cessation of growth prior to irradiation; vision normal; 10 yr. follow-up since irradiation
7. S. R. (non-hospitalized case)	Radiation therapy	Relief of headache; subsequent recurrence of headache, probably of non-pituitary origin; severe hypertensive; cerebral vascular accident
8. I. S. (non-hospitalized case)	Radiation therapy	Headache, somnolence improved; no further progression of acromegaly; 1½ yr. follow-up; vision normal
9. S. M. (non-hospitalized case)	Radiation therapy	Unimproved symptomatically; vision normal; advancing acromegaly; 3 yr. follow-up
10. V. K. (non-hospitalized case)	Radiation therapy	Cessation of growth; normal menses restored; vision normal; 4 yr. follow-up
11. M. A. (non-hospitalized case)	Radiation therapy	Bitemporal hemianopsia; maintained same vision; no further progression of acromegaly; improved symptomatically; 2½ yr. follow-up
12. R. B. (N. I. #22185-H)	Radiation therapy	Questionable slight bitemporal defect; relief of headache; no further progression of acromegaly; recent visual fields normal; 13 yr. follow-up
13. H. S. (non-hospitalized case)	Radiation therapy	Relieved of paresthesias; no further acral enlargement; 3 yr. follow-up
14. H. W. (non-hospitalized case)	Radiation therapy	Relieved of headache; potency better; no progression of acromegaly
15. S. D. (non-hospitalized case)	Radiation therapy	Symptomatically improved; no further acral enlargement; 3 yr. follow-up
16. J. J. (J. H. #262844)	Radiation therapy	Bitemporal hemianopsia; diplopia and weakness relieved; feels fine (letter); 3 yr. follow-up
17. I. F. (non-hospitalized case)	Radiation therapy	Relieved of headache and joint pains; 5½ yr. follow-up
18. E. P. (non-hospitalized case)	Radiation therapy	Headache relieved; hands softer and more pliable; 9 mo. follow-up
19. A. L. (N. I. #29442)	Radiation therapy	Bitemporal hemianopsia; relieved of headache and weakness; vision unchanged; 1 yr. follow-up
20. R. C. (J. H. #249469)	None	Homonymous hemianopsia; severe hypertension with hypertensive neuroretinopathy; death from cerebral hemorrhage
21. E. U. (non-hospitalized case)	Radiation therapy	Relieved of joint pains; no progression of acromegaly; feeling well; 9 yr. follow-up
22. M. Y. (non-hospitalized case)	Radiation therapy	Relatively asymptomatic for 12 yr.; recurrence of headache, drowsiness, further acral enlargement; vision unimpaired
23. P. W. (M. H. #27280-R)	Radiation therapy	Relieved of headache; no further progression of acromegaly; 11 yr. follow-up
24. C. Z. (non-hospitalized case)	Transphenoidal operation by Dr. Cushing in 1912	No headache since operation; no change in features for many years; normal vision; two children, aged 15 and 10; diabetic; 35 yr. follow-up

TABLE XII (*Continued*)

Case	Therapy	Course
25. M. A. (non-hospitalized case).....	Radiation therapy	Symptomatically improved; slight regression of acromegaly; 1 yr. follow-up
26. F. C. (non-hospitalized case).....	No indication for therapy	Asymptomatic; no recent progression of acromegaly; vision intact
27. B. S. (non-hospitalized case).....	Radiation therapy	Asymptomatic; questionable further growth of hands recently; further radiation therapy; 7 yr. follow-up
28. P. K. (J. H. #239283).....	Radiation therapy; operation	Persistence of headache despite irradiation; no visual disturbances; operation; postoperative recurrence of headache; further irradiation; relief of headache; improvement in features, return of menses; had a baby; 6 yr. follow-up
29. H. B. (M. H. #41602).....	Radiation therapy; operation	Headache and further acral growth despite irradiation; (6 courses over 9-year period); normal vision; operation; slight headache; 7 mo. postoperative
30. E. L. (M. H. #40150).....	Radiation therapy; operation	No relief of headache with irradiation; further acral enlargement; vision normal; operation; still having headache; 1 yr. postoperative; no increase in acromegalic features
31. M. G. (J. H. #241271).....	Radiation therapy; operation	No relief of headache with irradiation, nor following operation and postoperative radiotherapy; neurotic; no progress of acromegaly, nor visual impairment over 11 yr. follow-up period
32. P. W. (J. H. #266421).....	Radiation therapy; operation	Headache not relieved by irradiation; operation; improved; menses every 2 mo; vision normal.
33. J. R. (non-hospitalized case).....	Operation elsewhere via transphenoidal route; radiation therapy	Bitemporal hemianopsia; vision improved postoperatively; no evidence of further progress, 6 yr. after operation; headache due to associated hypertension
34. W. B. (non-hospitalized case).....	Radiation therapy; operation elsewhere	Bitemporal hemianopsia; visual impairment unrelieved by radiotherapy; operation; vision postoperatively about the same; feels fine 7 yr. postoperatively (letter)
35. M. M. (J. H. #272168).....	Two operations elsewhere; radiation therapy	Bitemporal hemianopsia; operation in 1933 because of visual impairment; recurrence of symptoms and reoperation in 1937; recurrence of visual symptoms and further acral growth in 1943; radiation therapy; no further progression of acromegaly; no further deterioration of vision; 3½ yr. follow-up; postoperative seizures

MALIGNANT ADENOMA—CASE REPORT

There was one instance of a malignant adenoma in this series.

G. O., Montefiore Hospital, No. 40337. An eighteen year old boy was first seen in this clinic on March 22, 1946, because of headache and diminished vision in the right eye. He had previously consulted a physician in 1944 at the age of sixteen because of failure to grow in the preceding four years. Physical examination at that time was essentially negative. He was 59 inches (147.5 cm.) in height. Basal metabolic rate was −11 per cent. Roentgen examination of the sella turcica was reputedly negative. Thyroid medication was prescribed. In January, 1945, he began to complain of headache relieved by vomiting. This headache continued intermittently and in January, 1946, an ophthalmologist was consulted who found evidence of optic atrophy. In the period of a year, the patient had gained 22 pounds and grown 1 inch. Roentgen study of the skull at this time revealed marked enlargement of the sella turcica with thinning of the posterior clinoids. There was no suprasellar calcification. Basal metabolic rate was −21 per cent. A glucose tolerance

test revealed a normal curve. The fasting blood sugar was 139 mg. per cent.

Examination in this clinic on March 22, 1946, revealed a short, pale, obese boy with a Froehlich type of body build. The genitalia were small, pubic and axillary hair scanty, and the hair on his head of a silk-like quality. Both optic discs showed considerable atrophy. Visual acuity was 15/30 in the right eye and 15/70 in the left eye, not improved by correction. Perimetric examination revealed constriction of the left visual field, with a depression in the upper temporal field, and a right temporal hemianopsia together with a defect in the right inferior nasal field. The clinical picture suggested a craniopharyngioma while the x-rays favored a pituitary adenoma or possibly an intrasellar craniopharyngioma.

On April 24, 1946, a right transfrontal craniotomy was performed. A cystic tumor medial to the right optic nerve was encountered and an incomplete removal accomplished. Examination of the tissue by Dr. H. M. Zimmerman revealed tumor cells of pituitary parenchymal origin showing pleomorphism and mitotic division. These cells invaded a thickened dural capsule. They appeared to be neither chromophilic nor ordinary chromophobe cells. The pathologic diagnosis was a pituitary carcinoma.

Unfortunately institution of radiation therapy was delayed. From November 29, 1946, to January 27, 1947, the patient received 1,000 r each to a right and left lateral portal and 825 r to a frontal portal. Visual loss has been progressive so that now vision in the right eye is limited to light perception. Visual acuity in the left eye is 15/20 but the field of vision has diminished considerably.

PITUITARY BASOPHILISM

Five patients were observed who appeared to fulfil the criteria for a diagnosis of Cushing's syndrome. Their ages were twenty-three, thirty, thirty-two, thirty-four and fifty-five years, respectively. Four of the five patients were females. All presented manifestations of the disease to varying degrees. The findings included adiposity of the face, neck and trunk (buffalo distribution), hirsutism, osteoporosis, sexual dystrophy, hypertension, purplish striae, polycythemia, kyphosis, a tendency to bruise easily, purpura, diminished resistance to cutaneous infection, acrocyanosis, edema of the lower extremities, slight exophthalmos and weakness.

A variety of therapeutic procedures were utilized. In the one case in which an adrenal tumor was found, this was removed. Irradiation of the pituitary was given a trial in three cases. In none did it appear to yield beneficial results. Radon seeds were implanted into the pituitary fossa of one patient. Although insufficient time has elapsed to evaluate fully the effects of this procedure, some improvement seems to have been achieved clinically. Two patients were also under the care of Dr. Fuller Albright who treated them with testosterone and potassium chloride. The testosterone was subsequently discontinued, and on the potassium chloride medication alone favorable results appear to have been obtained in one of these patients. Summaries of the five cases follow:

L. S., a thirty year old white male, a dentist by occupation, first came under observation on September 12, 1945, because of a multiplicity of symptoms. He was a known hypertensive since August, 1943. About that time, too, he had begun to develop a fulness in the region of his jowls and redness of the face. During the summer of 1945, he became aware of decreased libido though potency was retained. More recently he noticed some swelling of the lower extremities and a tendency to bruise easily. He gained 6 pounds and whereas his extremities remained unchanged, his face and abdomen had become more obese. In August, 1945, he developed large purple striae over the abdomen. There was also some diminution in the growth of his beard.

The significant findings on examination included a roundness of the face which was unusually florid, slight exophthalmos of the left eye, an acneiform eruption of the upper trunk, a slight buffalo hump, some pitting edema of both feet, purple striae over the lower abdomen and a blood pressure of 160/120 mm. mercury. The diagnosis of a Cushing syndrome was made and in December, 1945, he was referred to Dr. Fuller Albright at the Massachusetts General Hospital for further study.

There, detailed laboratory studies were performed, the results of which are listed below:

Hemoglobin	16.9 Gm./100 cc.
Red count	5.8 million
White count	14,400 (86% polymorphonuclear leukocytes, 10% small lymphocytes, 3% monocytes)
Serum calcium	10.6 mg./100 cc.
Serum phosphorus	3.2 mg./100 cc.
Serum phosphatase	3.2 Bodansky units
Vitamin A	1.2 units/cc.
Carotinoids	2.2 units/cc.
Cephalin flocculation	24 hours—negative; 48 hours—negative.
Serum cholesterol	212.0 mg./100 cc.
Van den Bergh	normal
Serum protein	6.96 Gm./100 cc.
Serum albumin	5.24 Gm./100 cc.
Serum globulin	1.72 Gm./100 cc.
A/G ratio	3.10
Serum N. P. N	27.0 mg./100 cc.
Serum sodium	145.9 mEq./L.
Serum potassium	3.9 mEq./L.
Serum chloride	105.0 mEq./L.
Serum CO_2 content	30.9 mEq./L.
Glucose tolerance test:	
Fasting	79 mg./100 cc.
½ hour	140 mg./100 cc.
1 hour	140 mg./100 cc.
2 hours	86 mg./100 cc.
3 hours	94 mg./100 cc.
4 hours	92 mg./100 cc.
Insulin tolerance test: (7.8 units insulin intravenously)	
0	103.0 mg./100 cc.
20 minutes	78.0 mg./100 cc.
30 minutes	57.0 mg./100 cc.
52 minutes	78.0 mg./100 cc.
60 minutes	75.0 mg./100 cc.
90 minutes	100.0 mg./100 c.
120 minutes	99.0 mg./100 c:.
45 minutes after 0.55 cc. of epinephrine 1/1000	132 mg.
60 minutes after epinephrine	144 mg.
Prothrombin time normal.	
Urine culture: alpha hemolytic streptococci and Staphylococcus albus	
Electrocardiogram normal	
17-ketosteroid excretion:	21.0 mg./24 hours
Beta ketosteroid	1.1 mg./24 hours
11-oxysteroid excretion	3.7 mg./24 hours
Intravenous pyelography revealed normal findings except that the left kidney was lower than the right.	

X-rays of the skull and spine were normal. The findings of an elevated 11-oxysteroid excretion, high serum sodium level, low potassium content, high white blood count and low percentage of lymphocytes were in keeping with the diagnosis of Cushing's syndrome. There was marked variation in the degree of abnormality of these findings. His 11- oxysteroid excretion varied from 3.7 to 0.27 mg./24 hours; 17- ketosteroid excretion varied from 29 mg. to 9.2 mg./24 hours; the percentage of lymphocytes varied from 22 to 9 per cent.

The urinary infection responded to 5 Gm. of sulfathiazole daily. Symptomatic improvement was obtained with potassium chloride in 5 Gm. daily doses.

Perirenal air insufflation was non-contributory. On December 22, 1945, Dr. Oliver Cope explored the left adrenal gland. No tumor was found and biopsy showed a normal or hyperplastic gland.

The laboratory indices reached their most abnormal values on the second postoperative day. Thereafter steady improvement occurred. Thus 11- oxysteroid excretion was 5.0 on December 23rd, 2.6 on December 24th, 3.0 on December 25th, 1.6 on December 26th, 0.4 on December 27th, 0.46 on January 11th, 0.43 on January 17th, 0.48 on January 29th, 0.42 on February 4th, and 0.34 on February 10th. The percentage of lymphocytes rose from 9 on December 24th to 35 on February 9th.

Irradiation of the pituitary was begun on December 28, 1945, and completed on January 11, 1946. He received 900 r to each of four portals.

Metabolic studies revealed a positive nitrogen balance but a negative calcium balance. The addition of potassium chloride further increased the positive nitrogen balance and decreased to one-third the negative calcium balance.

He was discharged from the hospital on February 10, 1946, on a regimen of five 0.3 Gm. capsules of potassium chloride. When reexamined in March, 1946, he showed considerable improvement. All his signs appeared to be regressing. Cyanosis was much less pronounced. The jowls and heavy fat deposits in the clavicular and breast regions were appreciably reduced. Striae were still present, though much paler. He felt stronger and libido was definitely improved. In July, 1946, his blood pressure was 170/90 mm. mercury. Further laboratory studies by Dr. Albright revealed the following data:

White blood count	9,900
Differential count	73% polymorphonuclear leukocytes, 20% small lymphocytes, 5% monocytes, 2% eosinophiles
Urine culture	Occasional alpha hemolytic streptococci and Staphylococcus albus

17—ketosteroid excretion	17.2 mg./24 hours
11—oxysteroid excretion.	1.1 mg./24 hours
Serum sodium	149.1 mEq./L.
Serum potassium	5.3 mEq./L.
Serum chloride	106.0 mEq./L.
Serum CO_2	32.3 mEq./L.
Serum sugar	91.0 mg./100 cc.
Serum cholesterol	287.0 mg./100 cc.
Total protein	7.06 Gm./100 cc.
Serum albumin	4.84 Gm./100 cc.
A/G ratio	2.2
Serum N. P. N	31.0 mg./100 c.

The normal white blood count and absence of a marked lymphocytopenia indicated improvement. The blood pressure, sodium level and 11- oxysteroid excretion were still abnormal. Another course of sulfathiazole was prescribed, as well as continuation of the potassium chloride.

In October, 1946, the patient reported further improvement. He had been playing tennis and doing a full day's work in the office. His blood pressure was 170/100 mm. mercury. He weighed 192½ pounds (197½ pounds in June, 1946). Urine culture still showed a few staphylococcus albus and non-hemolytic streptococci colonies. Laboratory studies revealed a diminution in the sodium and cholesterol content of the serum and a normal 11- oxysteroid excretion.

White blood count	13,000
Differential count	80% polymorphonuclear leukocytes, 16% small lymphocytes, 4% monocytes
Serum sodium	143.0 mEq./L.
Serum potassium	4.6 mEq./L.
Serum chloride	104.0 mEq./L.
Serum CO_2	31.0 mEq./L.
Serum cholesterol	231.0 mg./100 cc.
17—ketosteroid excretion	21.2 mg./24 hours
11—oxysteroid excretion.	0.4 mg./24 hours

In January, 1947, another course of sulfathiazole therapy was administered because of persistence of a positive urine culture. At that time serum sodium, potassium, chloride and CO_2 were normal. The 17- ketosteroid excretions were 18.8 and 26.6 mg./24 hours; the 11- oxysteroid excretions 0.48 and 0.56 mg./24 hours. Daily injections of 25 mg. of testosterone were administered between May 17th and May 30, 1947. No improvement resulted from this treatment.

At present the patient feels quite well. His weakness has not reappeared; libido and potency are normal; blood pressure is 150/90 mm. mercury. His face is less round and florid. Purplish striae are still evident. Visual fields are full and visual acuity normal bilaterally. He continues to take 4½ Gm. of potassium. chloride daily.

The most recent laboratory studies performed on May 31, 1947 revealed the following:

Serum sodium	140.0 mEq./L.
Serum potassium	4.5 mEq./L.
Serum chloride	103.0 mEq./L.
Serum CO_2	28.4 mEq./L.
Urine culture	Moderate growth of staphylococcus albus; diphtheroid and B. coli
17—ketosteroid excretion	11.6 mg./24 hours
11—oxysteroid excretion	0.65 mg./24 hours

B. C., Montefiore Hospital, No. 42543. A thirty-two year old white, Jewish female was referred to this clinic in March, 1947. She had been well till after the delivery of her second child in October, 1942, when her menses failed to return. Menstrual periods were resumed following estrogen therapy but ceased when this medication was discontinued. Some time in 1945 a redistribution of adipose tissue occurred so that her face, chest, abdomen and hips increased in size, whereas her arms and legs seemed thin in proportion. An increasing roundness and duskiness of her face was noted. There was no gain in weight, however. About this time, too, an elevated blood pressure was discovered. An increased growth of hair appeared over the upper lip and extremities. Her skin began to bruise easily and a marked pyodermia also developed. Weakness and fatiguability had become progressively more marked for a year and a half.

She was studied at another hospital where the diagnosis of Cushing's syndrome was made. There was no evidence of an ovarian or thymic lesion. Perirenal air insufflation was carried out, enlargement of the right adrenal gland suspected, and on July 31, 1946, an exploratory operation performed. No tumor was found but only what was thought to be a hyperplastic adrenal gland. Radiation therapy was instituted in November and December, 1946, and a total of 2,000 r administered to the pituitary region. No improvement resulted.

Examination in this clinic revealed a short, obese female with a rounded face and thin limbs. Her face and extremities were purplish-red in color and her chest covered with a maculopapular rash. There were ecchymoses and healed abrasions over both legs. A slight excess of hair involving especially the mustache area and the region about the nipples was

FIG. 18. Radon seeds are visible within the sella turcica; the latter is normal in size. There is a demineralization of the entire skull.

noted. No striae were seen. Blood pressure was 190/115 mm. mercury. Laboratory studies revealed the following:

Red blood count	5.0 million
White blood count	11,650 (normal differential count)
Platelet count	232,000
Bleeding time	2½ minutes
Clotting time	3 minutes, 5 seconds
Prothrombin time	
Undiluted plasma	22.9 seconds
Diluted plasma	49.6 seconds
Blood urea nitrogen	12.5 mg./100 cc.
Serum cholesterol	145 mg./100 cc.
Blood sugar	85 mg./100 cc.
Serum calcium	10.1 mg./100 cc.
Total protein	7.2 Gm./100 cc.
Albumin content	5.6 Gm./100 cc.
Globulin content	1.6 Gm./100 cc.
A/G ratio	3.8
Glucose tolerance test	
Fasting	85 mg./100 cc.
1 hour	296 mg./100 cc.
2 hours	206 mg./100 cc.
3 hours	49 mg./100 cc.
4 hours	66 mg./100 cc.
Basal metabolism	−16 per cent

Perimetric examination indicated no defects. Electroencephalography revealed an essentially normal pattern. The electrocardiogram showed a left axis deviation. Roentgen examination of the skull, spine and extremities indicated a moderate degree of demineralization; the sella turcica was not enlarged and there was no erosion of the clinoid processes.

Neoplastic disease of the adrenals having been excluded, a direct surgical attack on the pituitary gland was planned and on May 5, 1947, a right transfrontal craniotomy was performed. An anteriorly placed optic chiasm was visualized and it was immediately apparent that an attempt to incise the diaphragma sellae and remove tissue with a pituitary spoon would jeopardize the integrity of the chiasm. An opening was, therefore, made in the diaphragm with a lumbar puncture needle and five radon seeds, each containing 1.04 millicuries, implanted. (Fig. 18.) The patient withstood the operative procedure very well and her postoperative course was equally uneventful.

Since discharge from the hospital on June 2, 1947, the patient has been seen at frequent intervals. Subjectively she feels much improved. Objectively a few slight changes have occurred. Her dusky color is a trifle less marked and the contour of her face has tended to assume more normal proportions. Menses have not occurred. Her weight is unchanged over the preoperative figure (132 pounds). Blood pressure has varied between 135/85 and 170/110 mm. mercury.

Further observation will be necessary to evaluate the results of treatment in this case.

M. H., Montefiore Hospital, No. 41263. A fifty-five year old Jewish housewife was admitted on October 4, 1946, because of backache of several years' duration. Eight months ago a sensation of something snapping in her low back was experienced after bending, followed by a persistent, severe non-radiating pain. About two years previously, a change in her appearance had become apparent. She developed a puffiness of the face and neck and a prominence of the eyes. Her basal metabolism was found to be low and thyroid medication was administered. During the past year, she had been physically weaker. A splenectomy was performed twenty years ago for purpura hemorrhagica; purpuric spots were still evident occasionally.

She presented a striking appearance on physical examination. Her face was plethoric with heavy jowls, some telangiectases and a moderate degree of hirsutism on the sides of the cheeks and on the upper lip. Her extremities relative to her torso were thin. There were no striae present. The lumbosacral spine was tender and the Lasegue maneuver positive bilaterally. No neurologic abnormalities were demonstrable. Blood pressure was 115/85 mm. mercury. Laboratory data were as follows:

Red blood count	5.07 million
Hemoglobin	14.8 Gm./100 cc.
White blood count	9850 (75% polymorphonuclear leukocytes, 21% lymphocytes, 1% eosinophiles, 3% monocytes
Platelet count	268,000
Bleeding time	1½ minutes
Clotting time	2 minutes
Plasma prothrombin time	
Undiluted plasma	18 seconds
Diluted plasma	38 seconds
Blood sugar	112 mg./100 cc.
Blood urea nitrogen	15.8 mg./100 cc.
Serum cholesterol	279 mg./100 cc.
Serum chloride	343 mg./100 cc.
Serum calcium	10.6 mg./100 cc.
Serum sodium	331 mg./100 cc.
Serum phosphorus	3.4 mg./100 cc.
Alkaline phosphatase	3.9 units
Serum total protein	6.7 Gm./100 cc.
Albumin fraction	4.8 Gm./100 cc.
Globulin fraction	1.9 Gm./100 cc.
Glucose tolerance test	
Fasting	74 mg./100 cc.
½ hour	150 mg./100 cc.
1 hour	110 mg./100 cc.
2 hours	66 mg./110 cc.
Insulin tolerance curve	
Fasting	70 mg./100 cc.
20 minutes	41 mg./110 cc.
40 minutes	47 mg./100 cc.
60 minutes	161 mg./100 cc.
90 minutes	108 mg./100 cc.
45 minutes after adrenalin	155 mg./100 cc.
Urine examination	negative
Basal metabolism	−12 per cent
Sulkowitch test	positive
11—oxysteroid excretion	normal

Visual fields showed no defects. The electroencephalogram revealed an essentially normal pattern. Electrocardiography indicated a left axis deviation. Roentgenogram of the skull, thoracic cage and spine revealed a generalized osteoporosis; the sella turcica was otherwise normal.

Perirenal air injection revealed a distinctly enlarged adrenal gland on the right. The left adrenal gland could not be visualized on several occasions. It was, therefore, planned to explore the left side first and, if a normal adrenal gland was found, to remove the enlarged right one at a subsequent session. On January 29, 1947, the left suprarenal region was explored and an adrenal adenoma found and removed.

She continued to experience back pain postoperatively which was relieved by estrogen therapy. A thrombophlebitis of the left lower extremity followed by a pulmonary embolism complicated her subsequent course. After recovery from this complication, there was marked subjective improvement and she was able to get about without much pain. Prior to discharge from the hospital in August, 1947, her blood pressure ranged around 135/90 mm. mercury. Roentgenogram of her spine showed a collapse of the dorsal vertebrae 5, 6, 7 and 9. It was planned to fit her with a brace in the orthopedic clinic.

L. B., a thirty-four year old white female was seen on May 25, 1943, because of amenorrhea of about two years' duration and swelling of the face. An elevation in blood pressure had recently been discovered. She had always been inclined to have an excess of hair, perhaps more so lately. A tendency to bruise readily was also observed. She had had roentgenograms taken of her skull which were reported to be negative. Her basal metabolism was +2 per cent. She had been treated with thyroid extract, stilbesterol and aminophylline. The patient was married and had two children, one ten years old, the other six and one-half years of age.

Examination revealed a well developed female with a bloated face and jowls, facial hirsutism, prominence of the eyes, mottling of the skin, areas of ecchymoses, protuberance of the abdomen, slight edema of the feet and a buffalo hump. Blood pressure measured 190/130 mm. mercury. She weighed 110 pounds although she stated her usual weight fluctuated around 103 pounds.

Cushing's syndrome was diagnosed and radiation therapy to the pituitary instituted. Between June and October, 1943, three courses of irradiation, each totalling 2,400 r, were administered. The patient reported no change in her condition following this treatment.

She was subsequently under the care of Dr. Fuller Albright at the Massachusetts General Hospital during the spring and summer of 1946. Extensive studies were carried out, the results of which afforded confirmatory evidence of the diagnosis of Cushing's syndrome. Thus, low serum phosphorus and potassium levels, a high serum carbon dioxide content and an elevated 11-oxysteroid excretion were found. The insulin tolerance test was, however, normal. There was generalized osseous decalcification. The electrocardiogram indicated a left axis deviation. Treatment consisted of 25 mg. of testosterone proprionate every other day and 8 Gm. of potassium chloride daily. A Bacillus coli urinary infection responded to streptomycin. Perirenal air studies revealed no definite abnormalities. Exploration of the left adrenal gland revealed no tumor. Biopsy showed a normal or hyperplastic gland. It was deemed advisable to explore the right adrenal gland at a subsequent session but permission to do so was not granted. Despite the large intake of potassium, her serum potassium remained low.

The patient was seen again on September 25, 1947, at which time she was feeling quite well. Her weight was 109½ pounds. She still bruised easily and her physical findings remained unchanged. Blood pressure was 190/140 mm. mercury. Menses were reestablished in September, 1946, and had been regular in occurrence since then. She was still taking 6 Gm. of potassium chloride daily.

M. G., Jewish Hospital, No. 218322. The original example of the syndrome of pituitary basophilism described by Harvey Cushing in 1912 as a "polyglandular syndrome of painful obesity, hypertrichosis and amenorrhea with overdevelopment of secondary sex characteristics" and subsequently reclassified as a basophilic adenoma, came under the senior author's observation in 1939. Being of unusual interest from the historical as well as from a clinical standpoint, her history will be briefly reviewed and the observations recorded in 1939 added.

A twenty-three year old unmarried woman of Jewish extraction was admitted to the Johns Hopkins Hospital on December 29, 1910, because of amenorrhea since the age of sixteen, increasing obesity, headaches, nausea and vomiting. Her weight had increased from 112 to 137 pounds in the two years prior to admission. Other symptoms included pain in the eyes, insomnia, tinnitus, extreme dryness of the skin, frequent sore throats, dyspnea, palpitation, purpuric outbreaks, recurring nose bleeds and marked constipation with bleeding hemorrhoids. Growth of hair had appeared on the face. She had become increasingly round-shouldered. There was marked weakness and constant backache and epigastric pain. Examination revealed a short, kyphotic woman, with a round, dusky face which had an excess of hair. Adiposity affected the face, neck and trunk and relatively spared the extremities. The body and lower extremities were especially cyanotic and the skin bruised easily. There was considerable pigmentation. Skull films revealed a normal sella turcica. The red blood count was 5.3 million, the white count 12,000, and the hemoglobin 85 per cent. Systolic blood pressure averaged 185 mm. mercury. In September, 1911, a subtemporal decompression was performed because of continued headache.

She was again studied at the Peter Bent Brigham Hospital in 1913 because of further increase in weight (151 pounds). Her symptoms and general condition were essentially unchanged. Blood pressure averaged 180/110 mm. mercury. There was still a slight polycythemia, the red blood count being very slightly in excess of 5 million.

Late in 1913 menses were resumed, though irregularly. They continued until about the age of forty-six. In 1917, an operation for a renal calculus was performed at another hospital.

In November, 1922, she was readmitted to the Peter Bent Brigham Hospital. Her blood pressure ranged around 160/95 mm. mercury, the red blood count was 5.24 million, the basal metabolic rate was −9 per cent. Her general appearance remained unaltered, although there

had been some loss of weight. Skull films showed a generalized demineralization.

She came under Dr. Cushing's observation again in February, 1932, at which time many of her old stigmas were no longer evident. Her weight was 105 pounds and her blood pressure normal. She no longer bruised easily, her plethora was gone and her striae pale instead of purplish. The red blood count was 5.19 million and hemoglobin 111 per cent (Sahli). Her kyphosis was more pronounced.

In January, 1939, she was admitted to the Jewish Hospital of Brooklyn because of pain in the left flank radiating to the left lower quadrant. She had passed six or seven calculi in her urine during the previous ten years. Retrograde pyelography revealed no evidence of a stone, nor of other renal abnormalities. Physical examination showed slight facial hirsutism, a relaxed, pendulous abdomen, remains of the old striae, thin but not disproportionate extremities. She weighed 117 pounds; her blood pressure was 130/80 mm. mercury. Laboratory studies revealed the following:

Hemoglobin	88 per cent
Red count	5.0 million
White count	5500 (52% polymorphonuclear leukocytes, 43% lym phocytes, 2% monocytes, 3% eosinophiles)
Serum calcium	12 mg./100 cc.
Serum phosphorus	2.9, 3.2 mg./100 cc.
Serum phosphatase	4.8 units
Blood sugar	87 mg./100 cc.
Blood urea nitrogen	16.8 mg./100 cc.
Blood uric acid	5.6 mg./100 cc.
Phenol sulphonphthalien excretion	57.8% (2 hours)
Urine	Trace of albumin

COMMENTS

Diagnosis. The diagnosis of a pituitary tumor, either of the chromophobe or chromophile type, ordinarily offers little difficulty. As a rule, both varieties are diseases of adult life. Patients suffering from chromophobe adenomas are much more frequently encountered; and usually when they reach the neurosurgeon, the outstanding symptom is impairment of vision. Acromegalics, on the other hand, seek advice primarily because of headache and also because of the striking alterations of their features. Manifestations of hypopituitarism are usually evident in the chromophobe group and include such signs and symptoms as pallor and atrophy of the skin, loss of hair, depression of sexual function, gain in weight, a lowered basal metabolic rate and a tendency toward somnolence. The characteristic bodily changes produced by an eosinophilic adenoma render the diagnosis obvious at a glance in a well marked case. The head is large, the jaw prognathic, the nose prominent, the tongue thickened and the teeth widely separated. The extremities are large, the shoulders rounded and the hands spade-like and puffy. Among the numerous complaints are headache, amenorrhea, diminished libido and potency, paresthesias, excessive perspiration and joint pains. Diabetes mellitus is not an infrequent occurrence in this disease. Many other signs and symptoms may be present, as indicated by Davidoff[6] in a review of 100 cases of Dr. Cushing's series. Occasionally, signs of hypopituitarism coexist with evidence of preexisting hyperpituitarism. The term "fugitive acromegaly" has been applied by Bailey and Cushing[3] to such mixed cases.

Visual disturbances resulting from pressure on the optic chiasm and nerves are exceedingly common, especially in the group of chromophobe adenomas, and are of the utmost diagnostic importance. The demonstration of a bitemporal hemianopsia clearly indicates a lesion involving the optic chiasm. This is the most constant visual field disturbance produced by a pituitary tumor. For the detection of early visual field changes, the use of the tangent screen at a distance of two meters, together with small test objects, is far superior to ordinary perimetry. Usually the earliest indications of chiasmal compression are defects in the superior temporal quadrants. With further growth of the tumor, a complete bitemporal hemianopsia results; and if the process continues to remain unchecked, the inferior nasal fields are affected and finally vision fails altogether. Primary optic atrophy accompanies these changes. This is the usual sequence of events. Occasionally, however, a visual defect other

than a bitemporal hemianopsia is observed. Thus, these tumors may implicate the optic tract on one side, in which case a homonymous hemianopsia is produced. Should the optic chiasm happen to be located further posteriorly than usual, only one optic nerve may be involved and the symptoms be referable to but one eye.

Confirmatory roentgenographic evidence of an intrasellar tumor, consisting of enlargement and atrophy of the sella turcica, is present in the vast majority of cases of chromophobe adenoma. To a somewhat lesser extent this is also true of the eosinophilic tumors. The first structures to be involved are the dorsum sellae and posterior clinoid processes, which become decalcified. As the neoplasm expands, the process of demineralization involves the sellar floor and the fossa increases in depth and anteroposterior diameter, assuming the so-called "ballooned" appearance. Eventually the tuberculum sellae and anterior clinoid processes also become atrophic. In acromegaly, additional extrasellar changes are the rule. The calvarium is thickened, the paranasal sinuses enlarged and the mandible prominently overdeveloped.

In the relatively rare case of chromophobe adenoma in which the sella turcica remains normal in size, doubt may arise as to the nature of the lesion and additional information may be necessary through pneumencephalography. Films taken in the upright position to permit adequate visualization of the basal cisterns are particularly informative. Tumors arising from the pituitary body displace the cisterna interpeduncularis dorsally and either obliterate the cisterna chiasmatis entirely or else displace it rostrally. Deformities may also be noted in the hypothalamic portion of the third ventricle as well as in the floor of the frontal horns of the lateral ventricles.

The clinical manifestations of acromegaly are so striking that confusion in diagnosis is hardly likely to occur. Difficulties may arise, however, in distinguishing between a chromophobe adenoma and a number of other parasellar lesions, such as a craniopharyngioma, suprasellar meningioma, glioma of the optic chiasm, aneurysm of the circle of Willis and rarely an epidermoid, chordoma, tuberculoma or gumma. Craniopharyngiomas are primarily, though not exclusively, tumors of childhood, are frequently associated with adiposogenital dystrophy and papilledema in children and in about 80 per cent of cases show spotty calcification above the sella turcica. Gas studies are likely to reveal a deformity of the third ventricle, especially of the hypothalamic portion, obliteration of the cisterna interpeduncularis and a defect of the rostral end of the cisterna pontis. The suprasellar meningiomas occur largely in women past middle age, do not give rise to endocrine changes, are usually unaccompanied by enlargement of the sella turcica and may produce a hyperostosis overlying the tuberculum sellae. Pneumencephalography typically reveals displacement of the cephalic margin of the cisterna interpeduncularis caudally, together with a concave defect rostrally. Truncation of the frontal horns of the lateral ventricles or deformities of their inferior margins may also be evident. Gliomas of the optic chiasm and nerves give rise to symptoms usually during childhood and may be associated with manifestations of von Recklinghausen's disease. The findings on funduscopic examination are variable, as are the visual field changes. These tumors may undermine the anterior clinoid processes, thereby producing a J-shaped sella turcica. Enlargement of the optic foramina can often be demonstrated. The abnormalities observed in the pneumencephalogram consist of obliteration of the cisterna chiasmatis and a slight deformity of the cephalic border of the cisterna interpeduncularis. The diagnosis of an aneurysm of the circle of Willis is reasonably certain whenever a history of subarachnoid hemorrhage is elicited. The onset of a sudden ophthalmoplegia together with pain about the eye and forehead is also very suggestive of an aneurysm. Occasionally, the demonstration of a curvilinear streak of calcium

on the x-ray film offers a clue to its presence. Visualization by angiography will usually resolve all doubt.

The syndrome of pituitary basophilism (Cushing's syndrome) has become a well recognized clinical entity since Cushing's original description. It is a disease which affects women much more frequently than men. The manifestations of this syndrome are painful adiposity of the face, neck and trunk, kyphosis associated with osteoporosis and backache, and occasionally with compression fractures of the vertebrae, hypercalcinuria frequently with nephrolithiasis, amenorrhea in women and impotence in men, hirsutism without other evidence of virilism, hypertension, a thin, reddish skin, purplish striae, acrocyanosis, a tendency to bruise easily, purpura, diminished resistance to cutaneous infection, polycythemia, slight exophthalmos, edema of the lower extremities, weakness and hyperglycemia. According to Albright,[1] the significant laboratory findings are an unresponsiveness to an alimentary hyperglycemia, insulin resistance, and usually a moderate increase in 17— ketosteroid excretion. Recently an excessive output in the urine of 11— oxycorticosteroids has been demonstrated in patients with this condition.[20]

The underlying pathology of this disease has been succinctly epitomized by Albright:[1] "A patient with Cushing's syndrome at autopsy may or may not have a cancer or an adenoma of one adrenal cortex; one who does not have this may or may not have a basophile adenoma of the pituitary; one with a basophile adenoma of the pituitary may have hyperplasia of the adrenal cortices, and in the author's opinion, probably always would be found to have if the criteria for making the diagnosis and for determining hyperplasia were sufficiently accurate; finally, a patient who has neither a basophile adenoma nor a tumor of one adrenal cortex may have and probably always does have hyperplasia of the adrenal cortices."

Treatment. A considerable body of evidence exists to bear out the contention that whenever possible, radiation therapy is the method of choice in the treatment of pituitary tumors. This is generally conceded to be the case as far as the eosinophilic adenomas are concerned, but there still is a difference of opinion as regards the effects of roentgen therapy on the chromophobe tumors. Thus, it was only recently stated in a discussion on the radiation treatment of cerebral neoplasms that "chromophobe adenomas are no longer treated by radiotherapy for no conclusive evidence of their response to treatment has been obtained."[14]

In a monograph on pituitary tumors published in 1925, Dott and Bailey[7] stated: "To summarize the treatment of hypophyseal adenomata, we advocate a trial of x-ray therapy unless there is imminent danger of loss of vision, when a sellar decompression should be done forthwith. During the course of treatment, the progress of events should be carefully controlled by repeated perimetric examinations. If visual impairment becomes greater, a transphenoidal operation should be carried out, with partial removal of the tumor. Later, should symptoms referable to a lateral cranial extension of the tumor persist, a transfrontal operation should be undertaken. Following all these measures, x-ray treatment should be given in the hope of retarding the further growth of the tumor. They should at first be administered at intervals of three or four weeks, and later the treatment should be administered as the progress of the particular case demands."

The results of the roentgen treatment of five cases of pituitary adenomas, four of which were chromophobe tumors and one a chromophile tumor, were reported by Dyke and Gross[9] in 1931. Marked improvement of vision was achieved in three cases, while in the remaining two cases, there was no further loss of vision. In every case, headache was entirely relieved.

In 1936, Dyke and Hare[10] reviewed a series of sixty-three cases of pituitary tumor treated by irradiation. There were

twenty-five chromophile adenomas and thirty-eight chromophobe adenomas in this group. The effects on the visual fields and on visual acuity were emphasized. Of the thirty-eight chromophobe adenomas, 26.4 per cent showed moderate to marked improvement, 26.3 per cent were unchanged and 47.3 per cent became worse. Of the twenty-five acromegalics, 76 per cent showed an interruption in the progress of the disease or definite improvement; 24 per cent showed progression of symptoms despite treatment. Henderson[13] compared the data of Dyke and Hare pertaining to the chromophobe tumors with the statistics from Cushing's material. He pointed out that following operation, 68 per cent of the cases improved and 5.4 per cent became worse, whereas irradiation resulted in improvement in only 26.4 per cent, while 47.3 per cent continued to become worse. Dyke and Davidoff[8] subsequently indicated that Henderson's comparison was not altogether valid. The figures quoted by Henderson represented immediate results following operation, whereas the data submitted by Dyke and Hare represented late results, on the average of four years after therapy. It was further indicated that according to Henderson's figures, about 50 per cent of all patients operated upon showed evidence of a recurrence of the tumor. Seventy per cent of the recurrences appeared within two years and 95 per cent within five years of operation. The percentage of recurrences in Cushing's series, as reported by Henderson, was much less in those patients who received postoperative irradiation.

Sosman[19] (1937) expressed an opinion to the effect that following irradiation, "satisfactory remission of symptoms and signs will be obtained in over 50 per cent of patients with chromophobe adenomas, in 90 per cent of chromophil adenomas, and in the majority of basophil adenomas." The technic employed by Sosman in the treatment of pituitary tumors is very similar to that of Dyke and Davidoff.

Very favorable results in the treatment of twenty-five cases of pituitary tumor by irradiation were reported by Kerr and Cooper[16] in 1942. All but two cases were chromophobe adenomas. The results were divided into "excellent," "good" and "poor" depending upon whether there was (1) an increase in visual fields, loss of headache and general improvement in the patient's over-all condition, (2) arrest of the process with loss of headache and (3) no demonstrable good effects of the treatment. Excellent results were obtained in 56 per cent, good results in 20 per cent and poor results in 24 per cent. Kerr[15] has subsequently reported the results of a follow-up survey of these twenty-five patients and expanded the series by adding another twenty-six cases. Of the original twenty-five cases, one was proved not to have a pituitary tumor, thereby reducing the series to twenty-four cases. The results were excellent in 58.3 per cent, good in 20.8 per cent and poor in 20.8 per cent. Of the entire series of fifty cases, 58 per cent had excellent results, 12 per cent good results and 30 per cent poor results. The group was composed of thirty-seven chromophobe and eleven chromophile adenomas, one mixed tumor and one basophilic adenoma. Seventy-two per cent of the chromophile cases and 70 per cent of the chromophobes responded favorably to treatment. The single case of basophilism expired suddenly one week after the completion of therapy. It would appear from this study that favorable results which last for a period of six years are usually of a permanent nature. Kerr advises that all types of pituitary tumor be initially treated by irradiation. Continued decrease in the size of the visual fields two months after the cessation of treatment is considered to be an indication for operation. Kerr treats his patients with a large amount of radiation administered in one course. Four portals are utilized, two temporal, a frontal and a vertical. The physical factors are 200 KV, 1.95 mm. Cu HVL, 50 cm. distance and a 5 cm. cone. One hundred roentgens are given to one field the first day. If there is no reaction, two fields receive 100 r each the second day

and thereafter 200 r are administered to each of two fields per day. A total of 2,000 r measured in air is delivered to each field.

Of the fifty-nine cases of chromophobe adenoma treated with irradiation in this series, improvement was obtained in seventeen. Still another patient who showed regression five years after radiation therapy and improved again following further roentgen treatment raises the figure of cases improved to eighteen or 30.5 per cent of the total treated. Visual failure was halted in an additional ten patients, three of whom were operated upon nevertheless. The effect of radiation on the remaining seven patients can be justifiably considered beneficial since their vision was stabilized at a satisfactory level. Thus a total of twenty-five patients or 42.4 per cent were favorably affected by radiation therapy. Twenty-nine chromophile adenomas were treated with irradiation. Improvement resulted in eighteen or 62 per cent.

The accumulated evidence would appear to justify the practice of initially treating pituitary tumors with irradiation unless vision is dangerously close to amaurosis. The primary purpose of operating on a pituitary adenoma is not to remove the tumor in its entirety but to relieve pressure on the optic chiasm. It is technically impossible to remove the tumor completely; and even if total removal were feasible, it would be accomplished at the expense of complete destruction of the pituitary gland. That relief of pressure on the optic chiasm can be achieved by irradiation alone in an appreciable percentage of cases has been adequately demonstrated. The factor of operative mortality also bolsters the argument in favor of irradiation. Exclusive of recurrences, operation was performed in fifty-two cases of chromophobe adenoma, in five cases of chromophile adenoma and in one case of a pituitary malignancy. Operation was also carried out in a patient with Cushing's syndrome for the purpose of implanting radon seeds into the pituitary gland. There were eight postoperative deaths with a resultant mortality figure of 13.6 per cent. This operative mortality is perhaps higher than that reported from some other clinics and distinctly higher than that in Dr. Cushing's series. One thing must be emphasized and that is that Dr. Cushing, who did not believe in irradiation, selected for operation all cases in which a pituitary tumor was suspected, the earlier in the disease the better. According to our attitude, however, the earlier cases with fewer symptoms, better physical status and lesser degree of depression of pituitary function are selected for radiation therapy. Operation is reserved for the cases in the latter group which fail to respond to irradiation. Otherwise surgical treatment is applied to late cases with advanced local, as well as endocrinologic disturbances, extensions of the tumor into the intracranial cavity and bizarre primary locations of the tumor outside the pituitary fossa. Such cases are obviously greater surgical risks and would naturally carry a higher mortality.

Failure to achieve improvement with irradiation cannot be attributed to cystic changes within the tumor alone. Of twenty-three cases of chromophobe adenoma unimproved or worse following roentgen therapy and operated upon, cystic tumors were found in only five. Variations in the degree of sensitivity to the effects of roentgen rays are probably a more common cause for the refractory behavior of many tumors.

Twenty-eight patients with chromophobe adenomas were subjected to surgery following radiation therapy. Operation followed by roentgen therapy in all but six cases (four postoperative deaths) was performed in another twenty-three patients. Of a total of fifty-one patients operated upon, thirty-four were improved. Four patients were unimproved, in each case vision prior to operation having been exceedingly poor. There were nine instances of recurrence. The treatment of recurrences followed the same plan advocated for cases receiving initial therapy.

Five patients with acromegaly were treated surgically. Improvement resulted in two of them. Two others continued to complain of headaches. The remaining patient was operated upon only recently.

Since the syndrome of pituitary basophilism may be associated with an adrenal tumor, diagnostic procedures to investigate this possibility must be instituted. Pyelography and perirenal air insufflation are routine procedures and even if they should provide negative evidence, exploration may still be advisable. Having excluded an adrenal tumor, irradiation to the pituitary may be attempted. The evidence in favor of this form of therapy is not especially convincing. No definitely beneficial results were noted in the three cases recorded by Dyke and Davidoff.[8] Eisenhardt and Thompson[11] reported temporary improvement in only one of eighteen patients treated with irradiation. Favorable results have been reported by Cushing[5] and Sosman[19] and recently by Ellman and Vilvandré[12] and by Luft.[17] No obvious improvement was noted in the three cases in the authors' series. On the assumption that a state of hyperpituitarism exists in Cushing's syndrome, Pattison and Swan[18] implanted radon seeds into the sella turcica of two patients with this disease who had been treated ineffectually with radiotherapy. Considerable improvement resulted though it was obvious that the disease had not been cured. This form of treatment was employed in one case in the present series. Insufficient time has elapsed to evaluate the result in unequivocal terms, but some clinical improvement at least appears to have been achieved thusfar. Albright[1,2] has suggested that testosterone and potassium chloride may be valuable adjuncts in the treatment of this condition.

The authors owe sincerest gratitude to Dr. Max Chamlin and Dr. Edwin Billet of the Ophthalmological Service of Montefiore Hospital for innumerable follow-up visual field studies. We also wish to thank Dr. Fuller Albright for permission to include laboratory data obtained in his clinic and Dr. H. Dabney Kerr for permitting us to read his paper presented before the American Roentgen Ray Society, in manuscript form before its appearance in print.

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LATERAL RUPTURE OF CERVICAL INTERVERTEBRAL DISCS

INCIDENCE AND CLINICAL VARIETIES

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LATERAL rupture of the intervertebral discs in the cervical spine is common. Symptoms are, as a rule, mild and do not require surgical intervention; total disability from this lesion is seldom encountered. The actual frequency of ruptures in this region is difficult to determine.

An idea of their relative incidence, however, may be gained from several sources; the x-ray department of a general hospital supplies considerable data. During a six months' period (January to June, 1947) a total of 5,557 patients was examined in the x-ray department of the Baptist Memorial Hospital This group included patients from the hospital, from the outpatient department and from the adjoining medical office building. In sixty-nine patients, or 1.2 per cent, x-rays showed changes characteristic of a ruptured cervical disc. In a number of instances, x-rays were requested of the shoulder and teeth; the clinical data furnished with the request indicated pain in the shoulder and arm, associated with a stiff and painful neck. Additional films were taken of the cervical spine; the source of trouble in most instances was found to be in the discs.

Data was obtained from a second source, a medical clinic (Lyle Motley). In 758 patients (March to September, 1947) a diagnosis of ruptured cervical intervertebral disc was made in thirty-three instances, or 4.3 per cent. While the diagnosis was not confirmed by surgery or postmortem examination in any patient, the classical x-ray and clinical findings were present in all.

Such a high percentage in this group is surprising. These were patients referred for diagnostic study, many of them suffering with pain in the precordium, supposedly of cardiac origin.

In the practice of neurosurgery one would expect this condition to be encountered more frequently. From our own records during the past six months (January through June, 1947) a diagnosis of ruptured cervical disc was made forty-nine times, or 1.9 per cent of 2,637 patients. All of the patients from this source who complained of pain in the occiput, neck, shoulder, arm and fingers were asked the routine questions and a routine examination was made for ruptured cervical disc. If the clinical picture was compatible with this condition, routine x-ray examination of the cervical spine was made. The diagnosis was verified at operation in eight patients in whom the severity and duration of the symptoms and the failure of conservative measures led to the recommendation and acceptance of surgical treatment. In the other forty-one patients, the diagnosis was based upon a characteristic history, typical findings, x-ray evidence and therapeutic tests. There were no deaths, consequently the diagnosis was not confirmed by postmorten examination in any of the patients.

The certainty of the diagnosis in some of these patients may be questioned but, on the other hand, in spite of the interest in these lesions in each organization, some cervical disc ruptures probably were overlooked. At any rate, there can be little doubt that rupture of cervical discs is responsible for a large number of occipital headaches, stiff and painful necks, shoulder and arm pains extending into the hands and fingers and anginalike pain. These statistics, although covering a short period and obtained from only three sources, indicate that this condition is sufficiently common to play an important part in diagnostic

roentgenology and diagnostic medicine as well as in orthopedic surgery and neurosurgery.

There are two groups which are clinically and pathologically different; the acute variety is characterized by sudden onset following definite trauma such as jerking of the neck. The pain is intolerable, the neck is stiff and nerve root involvement is evident from the sensory and reflex changes. Even weakness of the muscles and vasomotor disturbances are not infrequent. X-ray findings are limited to loss or reversal of the normal curve since the amount of displaced cartilage is not sufficient to result in narrowing of the interspace, nor has sufficient time elapsed for degenerative changes or bone reaction. In this group relief is not obtained by rest or traction and early operation is demanded by the excruciating pain. Operative findings in patients exhibiting this clinical picture are flattening and compression of the nerve root. The root is not adherent but is exquisitely tender; a small nodule of fibrocartilage bulges beneath the stretched and thinned posterior ligament in the manner usually found in the lumbar region. Following removal of the small mass of fibrocartilage, the nerve returns to its rounded contour. Relief is immediate and dramatic and the function of the neck and affected nerve is usually restored promptly.

The second group is the chronic type following mild or repeated trauma; the patient may not have any recollection of injury. At first there are cricks in the neck, then pain in the occiput, shoulder, arm, fingers and precordial region and numbness and tingling of the fingers. It is in this group that neuritis, tendonitis, bursitis, myositis, fibrositis, angina and the scalenus anticus syndrome are usually thought to be responsible for the symptoms. These patients are able to raise the affected arm over the head without discomfort. The pain is increased by downward pressure on the head and flexion of the neck and is relieved by traction on the head. Tenderness is elicitied by pressure on the rhomboid and the subclavicular regions and over the affected nerve trunk in the neck. Sensory and reflex changes may be present and atrophy of the affected muscles evident. In this group x-rays show, in addition to the loss or reversal of curve, narrowing of the affected interspace, or interspaces and osteophytes; the latter can usually be demonstrated in oblique views, extending into the neural foramen laterally so as to impinge upon the nerve root.

In the chronic group, surgery is indicated when severe pain and disability of long duration or of frequent recurrence has failed to respond to conservative measures such as bed rest and head traction, followed by the wearing of a suitable collar or support. The operative findings are very different from the acute variety. The nerve root is found to be flattened, adherent and compressed by a ledge of hard material narrowing the neural foramen laterally. The nerve root is squeezed so tightly in its foramen that even a small probe can be passed beside it with difficulty, if at all. The foramen is never foreshortened sufficiently in its longitudinal diameter to compress the nerve root, regardless of the degree of narrowing of the interspace. This may readily be proven by examining a skeleton with no padding whatever between the vertebral bodies.

In recent cases, the nerve root can be displaced either upward or downward with reasonable ease and the nodule of cartilage removed. In the chronic variety, it is more difficult to displace the nerve; removal of the hard material cannot be accomplished without risk of damaging the nerve. In some instances the calcified protrusions are sharp; excision with a chisel, curet or dental bur is indicated. Ordinarily, nerve root compression can be relieved by enlarging the neural foramen laterally at the expense of the facets.

Neither of these types seems to be related to the "explosive" central ruptures found in patients injured in diving accidents and violent automobile accidents; these were formerly attributed to "subluxation." Nei-

ther are they apparently related to the central ruptures which protrude slowly, simulating spinal cord tumors.

Lateral rupture of a cervical intervertebral disc, although very common and the cause of prolonged and recurrent symptoms, seldom causes incapacity. Surprisingly few of these ruptures are severe enough to require surgery for relief; most of these are of the acute variety with soft ruptures. Doubtless, many of the milder syndromes, with only cricks and stiffness in the neck and transient radiating pain, recover spontaneously without recognition.

Grateful acknowledgment is made to the Baptist Memorial Hospital and Lyle Motley Clinic for access to their records.

In neurosurgery, as in other fields, the most important diagnostic factors are the careful history and physical examination. Valuable adjuncts, such as perimetry, audiometry, roentgenography, electroencephalography, and cerebrospinal-fluid studies, may be essential.

RUPTURED INTERVERTEBRAL DISCS IN THE LOWER LUMBAR REGIONS*

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THE disc controversy continues unabated fourteen years after the classic description of the disorder by Mixter and Barr.[1] Unfortunately, the controversy is not confined to treatment methods but includes pathologic and clinical diagnostic problems as well.

In Mixter and Barr's original article this disorder was named "rupture of the intervertebral disc with involvement of the spinal canal." In a later paper Mixter and Ayer used the term "herniation or rupture of the intervertebral disc into the spinal canal."[2] These terminologies were an attempt to state accurately the exact anatomic and pathologic conditions found in the diseased discs. In 1934 Peet and Echols called the disorder "herniation of the nucleus pulposus."[3] Later, the term "protruded intervertebral disc"[4] was introduced into the literature. This term had broader implications than the ones of Mixter et al., for it included those discs that protruded backward into the spinal canal without frank rupture of the annulus fibrosus.

Dandy[5] proposed a new terminology—a "hidden" or "concealed" disc. His description applied more to a condition found at the operating table than to an actual pathologic state demonstrated on postmortem material. Later he introduced an entirely new conception into disc surgery, i.e., a large percentage of patients suffering from disc symptoms have multiple ruptured discs.[6]

In the beginning the neurosurgeons pioneered the problem; soon the orthopedic surgeons became interested in it. Their points of view were usually quite dissimilar to those of the neurosurgeons, for they considered a spinal fusion operation of one type or another to be a paramount consideration. More recently the general surgeons, particularly the industrial surgeons, have joined the ranks of disc enthusiasts. At present the patient with low back and leg pain is apt to receive about as many opinions as doctors whom he has consulted about his complaints. It is small wonder that reports of distressingly bad results[7] are creeping into the literature.

Our purposes in this report are: (1) To restate and clarify the fundamentals of the problem, i.e., anatomic, physiologic and pathologic features; (2) to re-evaluate the accuracy of the neurologic symptoms and signs; (3) to clarify the treatment methods based upon twelve years of active interest and experience and (4) to present a sample follow-up study of our results.

Anatomic Considerations. A thorough knowledge of the anatomic relations in the lower lumbar portion of the spine is essential to a clear understanding of the symptoms and signs of ruptured intervertebral discs.

Each intervertebral disc is composed of three parts: (1) annulus fibrosus, (2) nucleus pulposus, and (3) cartilaginous plates. (Fig. 1.)

The *annulus fibrosus* forms the periphery of the disc and is composed of concentric layers of fibrous and elastic tissue. Anteriorly, the annulus is thick and well defined and its fibers are blended with the strong anterior longitudinal ligament. Posteriorly, the annulus is about one-half the thickness of that on the anterior surface and is separated from the spinal canal by the

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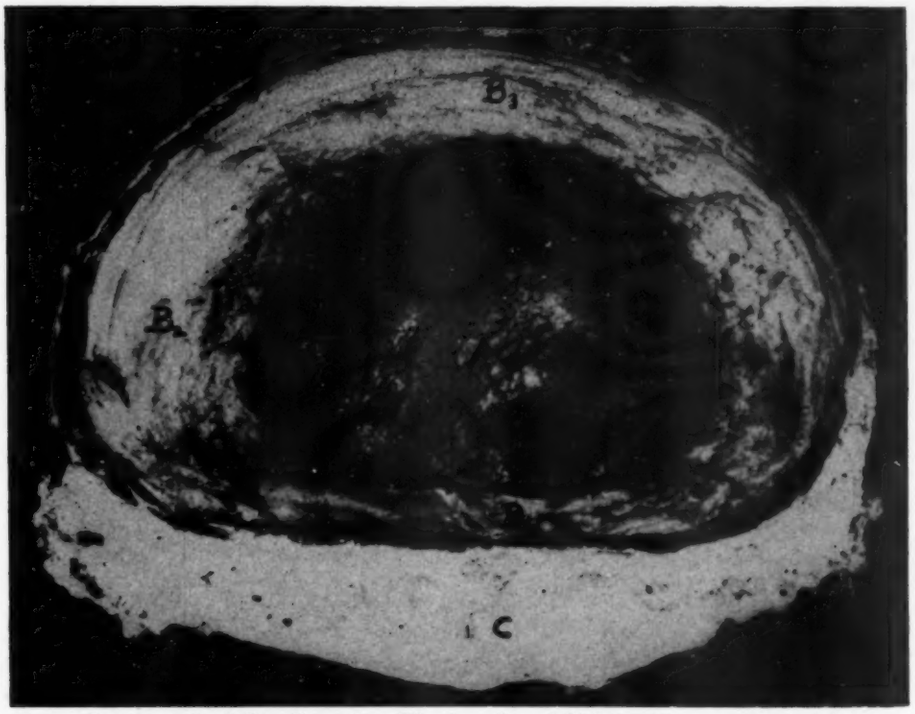


FIG. 1. Intervertebral disc dissection. The fourth and fifth lumbar vertebrae with intervening disc were removed intact from a fresh autopsy specimen. The intervertebral disc was split by cutting through the mid-portion of the annulus fibrosus, thus leaving two halves of the disc with smooth surfaces. The nucleus pulposus was carefully dissected from each half and the two parts glued together. Then the whole nucleus was covered with modeling wax. The nucleus and the depression from which half of it had been dissected were painted yellow. The remainder of the disc and the fifth lumbar vertebra were painted white. The nucleus was placed in its bed and the specimen photographed on panchromatic film. A, nucleus pulposus; B_1, B_2 and B_3, annulus fibrosus; C, body of the fifth vertebra.

posterior longitudinal ligament which is thin and weak, giving no support to the annulus except in the midline where it becomes somewhat thickened.

The *nucleus pulposus* occupies the approximate center of the disc. The point at which the annulus ends and the nucleus pulposus begins is not sharply defined. However, the main nuclear mass is quite distinct grossly from the remaining disc material. The nucleus pulposus is soft and has a semi-fluid consistency, particularly during the first three decades of life. Its water content is gradually reduced with age, until in the fourth and fifth decades it loses much of its elasticity. Histologically, the nucleus pulposus is composed of loose fibrous tissue interspersed with cartilage cells.

The *cartilaginous plates* separate the annulus and the nucleus from the adjacent vertebrae. The layer of cartilage is well defined in its central portion where it protects the adjacent vertebrae but thins toward the periphery where it blends with the annulus fibrosus.

Until recent years it was believed that there were no sensory nerves in the posterior annulus fibrosus. It has now been amply demonstrated that the annulus fibrosus is richly supplied with sensory nerves.[8] This anatomic fact confirms the clinical observations that manipulation of the annulus fibrosus in patients operated upon under local anesthesia causes severe low back pain.

The relation of the fifth lumbar nerve to the disc between the bodies of the fourth and fifth lumbar vertebrae and the relation of the first sacral nerve to the lumbo-

sacral disc are especially important, since over 98 per cent of all ruptured lumbar discs occur at these levels.

The spinal cord terminates opposite the intervertebral disc below the first lumbar vertebra. The nerve roots of the cauda

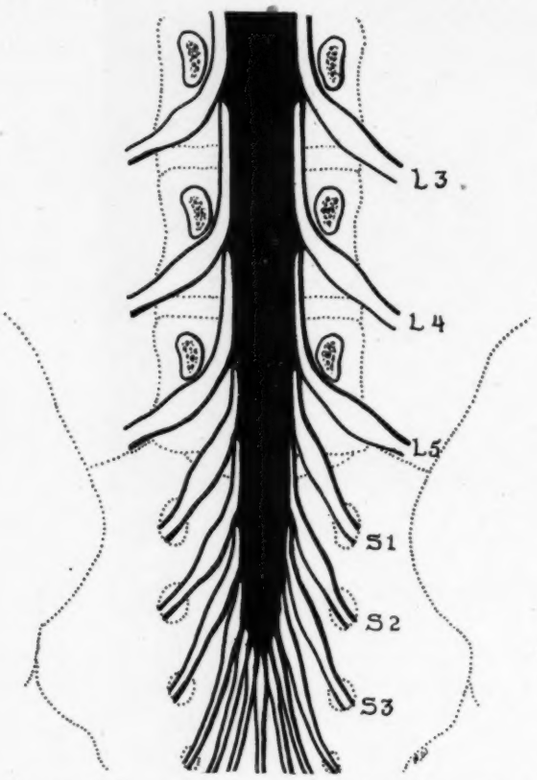


FIG. 2. Diagrammatic representation of the thecal sac and the lower lumbar and sacral nerves to the pedicles and intervertebral discs of the lower part of the spine.

equina are freely mobile in the large lumbar canal except as they approach their points of exit where they become fixed. (Fig. 2.) It is, therefore, apparent that only those roots that are compressed near their points of exit cause symptoms and signs of localizing value unless the lesion is a tremendously large one which uniformly compresses the roots of the cauda equina. At the level of the fourth lumbar disc the fifth lumbar nerve is contained in the subarachnoid space, fixed laterally against the dura one vertebra higher than its exit. The dural sleeve of the fifth lumbar nerve emerges below the fourth lumbar disc and passes downward beneath the pedicle of the fifth lumbar vertebra through its intervertebral foramen. (Fig. 3.) This relation holds between all the lumbar nerves and their respective discs. In contrast, the dural sleeve of the first sacral nerve separates from the thecal sac above the lumbosacral

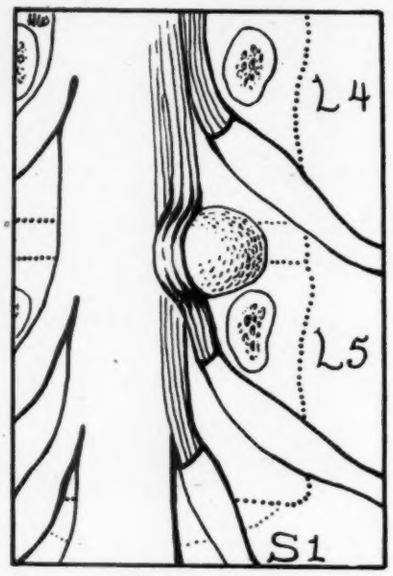


FIG. 3. Diagram illustrating the mechanism of compression of the roots of the fifth lumbar and first sacral nerves from a lateral herniation of the nucleus pulposus at the fourth lumbar interspace.

disc and can thus be compressed without deforming the sac.

It is necessary that any physician who wishes to localize a ruptured intervertebral disc on the basis of clinical findings has a thorough anatomic knowledge of the distribution of the fourth and fifth lumbar roots and the first sacral root. The sensory dermatomes of the fourth and fifth lumbar and the first sacral nerve roots are complex and a thorough knowledge of their complexity need not be discussed at this time because only certain portions of the dermatomes are essential for accurate diagnosis. As an example, the patch of skin on the medial aspect of the foot and leg just above the ankle represents the distal portion of the sensory dermatome of the fourth lumbar nerve, whereas, the area of skin on the dorsal surface of the great toe and a band extending directly upward on the

foot from the great toe is the distal distribution of the fifth lumbar dermatome. The first sacral dermatome supplies the skin on the lateral aspect of the foot, the outer two toes and perhaps the third toe as well. The dermatome extends over the lateral aspect of the ankle up the lateral aspect of the leg to just below the knee. (Fig. 4.)

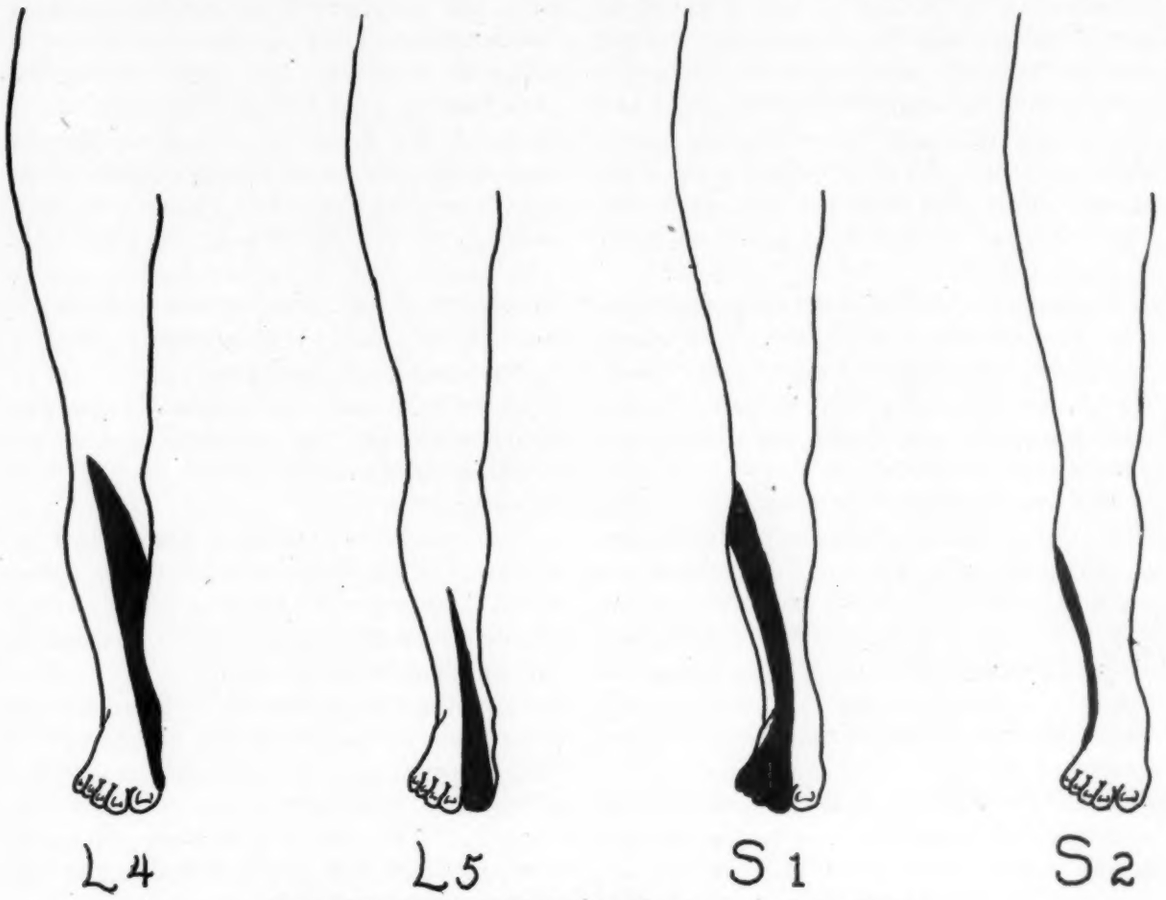


Fig. 4. Distal sensory distribution of L4, L5, S1 and S2 nerves.

The motor innervation of the two lower lumbar and first sacral nerves has considerable importance in diagnosis and localization. However, all of the muscles of the lower extremity have a dual or even a multiple segmental innervation so that paralysis of a muscle or muscle group is seldom seen in disc lesions. In the great majority of disc lesions only a single root is affected; hence the muscle or muscular group supplied by it would show only a minor degree of weakness.

In lesions affecting the fourth lumbar nerve (L3 disc) the quadriceps femoris is most affected. With involvement of the fifth lumbar nerve (L4 disc) the anterior tibial group particularly the extensor of the great toe may show evidence of weakness. In lesions of the first sacral nerve (L5 disc) the posterior calf muscles (gastrocnemius and soleus) are most often affected and weakness in these muscles may be easily demonstrated by having the patient walk on his toes. If weakness is present, the heel of the affected side will drop to the floor or at least show a tendency to do so.

The tendon reflexes are of great importance in diagnosis. If the knee jerk is depressed, it points definitely to involvement of the fourth lumbar nerve, i.e., L3 disc. An absent ankle jerk indicates a lesion of the first or first and second sacral nerves, i.e. L5 disc.

There are no very reliable reflex changes associated with a fourth lumbar disc lesion. As a matter of fact, given a patient with a characteristic history and with physical findings of a ruptured disc, who has normal knee and ankle jerks, the presumptive evidence is that the lesion is at the fourth lumbar disc. The posterior tibial muscle receives a large portion of its motor innervation from the fifth lumbar nerve and its tendon reflex may be abolished with lesions at this level. However, we have found this reflex difficult to elicit consistently and we doubt its practical value in diagnosis.

Physiologic and Pathologic Considerations. The intervertebral discs have frequently been compared functionally to a hydraulic shock absorber. Certainly, the structure of the intact disc would indicate that such a comparison is justified.

The misstatement is frequently made that the nucleus pulposus is highly expansile. Actually, the nucleus pulposus is incompressible and due to its plastic nature obeys the laws of fluids. It is the tension of the annulus fibrosus which keeps the nucleus pulposus under pressure, even when the intervertebral disc is not bearing weight.

The annulus fibrosus can be compared to a strong but somewhat elastic membrane firmly binding the vertebral bodies together. The nucleus pulposus which fills the cavity separating the vertebral bodies can be compared to a fluid. Although the nucleus pulposus is plastic rather than fluid, it behaves as a fluid in response to the limited degree of displacement to which it is subjected in movements of the vertebral column.[9] Barr's experiments are most illuminating in this regard.[10] He found that by compressing the vertebral column in a vice he could produce marked distention of the posterior annulus fibrosus. When this pressure was released, the annulus fibrosus returned to a normal shape. However, if he pierced the annulus fibrosus with a needle while the disc was compressed, the annulus fibrosus ruptured where it was penetrated, allowing the nucleus pulposus to extrude itself outward. When this occurred, the intervertebral disc lost its elasticity and failed to return to its former state.

When injury to the posterior annulus fibrosus or when the laminae of hyalin cartilage has allowed the nucleus pulposus to escape, the annulus fibrosus is subjected to forces which are alien to it. It becomes then merely a washer resisting direct force. With the fluid medium for even distribution of the transmitted force gone, movements are forced to bear upon a small part of the intervertebral disc at any given time. The disc is then in a position to be ground between the adjacent vertebral bodies. If the vertebral body is weakened by disease, it, too, may be ground away; at least it will become sclerosed and probably produce spurs to resist the unusual and poorly distributed force to which it has been subjected.

It is obvious, therefore, that when the anatomy of the disc is altered, repair by the usual processes may occur; but these reparative processes cannot hope to restore the normal physiology of the disc.

Carrying this reasoning into clinical application, it may be stated unequivocally that once a disc is ruptured *no surgical effort can hope to restore its anatomic integrity*. The natural healing processes may close the tear in the annulus fibrosus, or may even cause fibrous union of the contiguous cartilaginous plates, but nothing remotely resembling the normal shock-absorbtive mechanism can be restored. This reasoning applies with equal force to the disc that has been ruptured accidentally as well as to the normal disc that the surgeon who, through lack of knowledge or carelessness, has opened at the operating table in an attempt to find the diseased one.

We do not pretend to know how many different types of disc disorders may enter into the painful back syndrome. We do know that the posterior longitudinal ligament and the posterior annulus fibrosus are richly supplied with sensory nerve endings.

We know that manipulation of these structures in a conscious patient at the operating table produces the characteristic type of low back pain usually called "lumbago." But further than this we have no exact knowledge on this subject.

We are, however, thoroughly convinced that we know the type of disc disturbance which is responsible for *unilateral sciatic pain* associated with low back disability. The lesion that produces these disabilities is primarily one of the *posterior annulus fibrosus*. As we have previously indicated, there is a constant bulging of the posterior annulus fibrosus under conditions of stress and strain. However, in the intact disc this bulge disappears immediately once stress is removed, and the annulus fibrosus again assumes its normal contour. But when the annulus fibrosus is torn, the normal pressure relationships within the disc are immediately changed. If the tear in the annulus fibrosus involves the entire structure, there may be immediate herniation of the nucleus pulposus through the tear, thus producing a bulging mass within the spinal canal. The nucleus pulposus may even break through the flimsy posterior longitudinal ligament and lie loose within the spinal canal. A hernia thus produced is obviously an irreducible one; and no matter how much conservative treatment is used, such a herniation will not be relieved by natural healing processes. The patient, however, may eventually be relieved of his symptoms by total destruction of the nerve root.

The tear in the annulus fibrosus, however, does not necessarily have to be a complete one. The tear may involve only the inner group of fibers of the posterior annulus fibrosus and, therefore, create nothing but a weakened spot in the structure. However, as one would expect with any structure in which hydrodynamic forces are called into play, that spot being the weakest will bulge when strain is applied; in fact, it will bear the brunt of all changes in pressure relationships due to motion. (Fig. 5.)

The first type of disc disorder just described is the one which is so easily demonstrated at the operating table. The second type is quite a different matter, for here one may see only a localized bulge in the annulus fibrosus, or for that matter in certain positions in the recumbent position a bulge may not even be demonstrated. However, if one palpates the disc carefully with a blunt instrument the weakened spot can usually be identified. It may occur in any position from the midline to the most lateral limit of the posterior annulus fibrosus.

The first type of disc condition (frank rupture with herniation of the nucleus pulposus) described is the type that is refractory to conservative treatment. The second type (with incomplete rupture) may be more favorable for conservative therapy. We are quite convinced that many cases of the latter type recover spontaneously or with ordinary conservative treatment if this is instituted during the first attack of pain.

Clinical Considerations. In June, 1939, one of us (R. G .S.) in collaboration with Bradford[11] suggested a group of criteria that could be relied upon in the clinical diagnosis of ruptured intervertebral disc at either the fourth or the fifth lumbar interspace. These criteria were based upon a series of ten cases in which surgical procedures were done without the aid of contrast myelography.

In March, 1940,[12] we made a report upon the clinical diagnosis of intervertebral disc lesions in the lower lumbar region based upon experience with 125 consecutive operations. We found that the criteria originally presented by Spurling and Bradford are, on the whole, accurate. In addition, we presented the localizing features of ruptured intervertebral disc at the level of the third interspace.

Now, after seven years of additional experience we shall attempt to select the most important features of the subjective and objective examination in making the

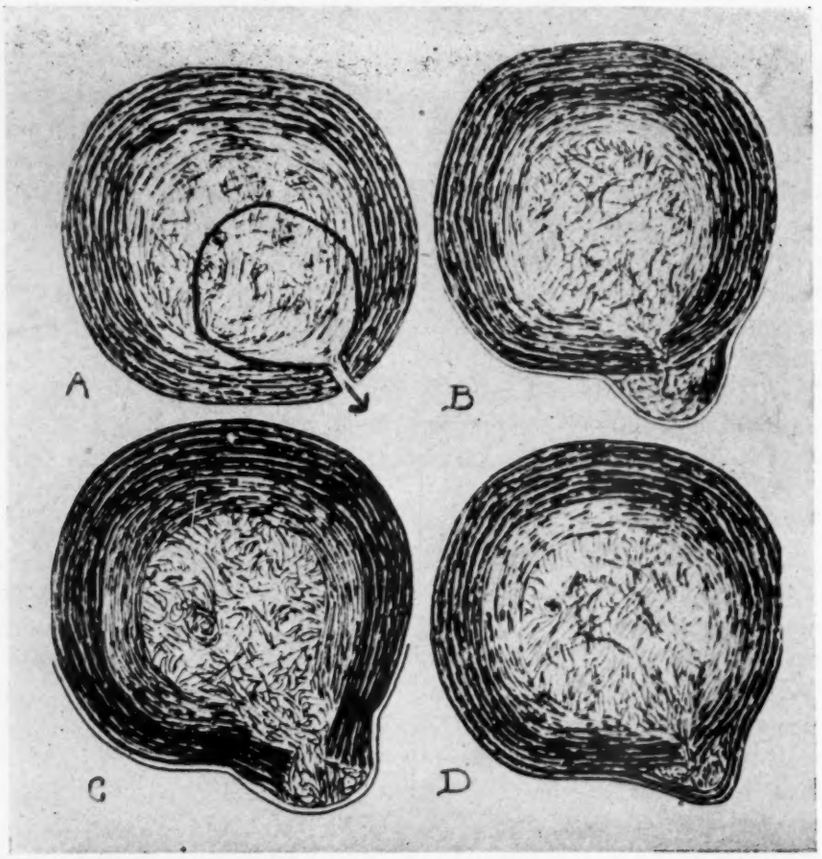


FIG. 5. The intervertebral disc is diagrammatically represented in cross section. The outer lamellae of the annulus fibrosus are represented as dense parallel bundles, the inner lamellae as lighter bundles and the nucleus pulposus as interlacing fibers. A, a line has been drawn around that portion of nucleus pulposus and inner lamellae of the annulus fibrosus which will rupture through the defect in the outer lamellae of the annulus fibrosus. B, the herniation is completed. C, a rarer type of herniation in which the outer annulus fibrosus is fractured. D, an interlamellar herniation in which the two outermost lamellae of the annulus fibrosus have distended over the mass without rupturing.

clinical diagnosis of ruptured intervertebral disc and also in accurately localizing it.

HISTORY

With rare exceptions, the first symptom of a ruptured intervertebral disc is backache. Direct trauma to the spine is not a factor in more than 40 per cent of the cases. A frequent story is that when in a bent forward position, lifting a heavy object, the patient felt a sudden catch in the back and straightening upright could be accomplished only with excruciating pain. The outstanding characteristic of the backache is its mechanical nature. It is intensified by any movement, particularly on bending forward or straightening upright. Twisting motions, such as turning over in bed, are especially painful. Relief of the pain is usually accomplished by lying perfectly still, although some patients cannot tolerate the supine position and can find comfort only in a sitting or even a jackknife position. The pain is usually intensified by coughing, straining or sneezing.

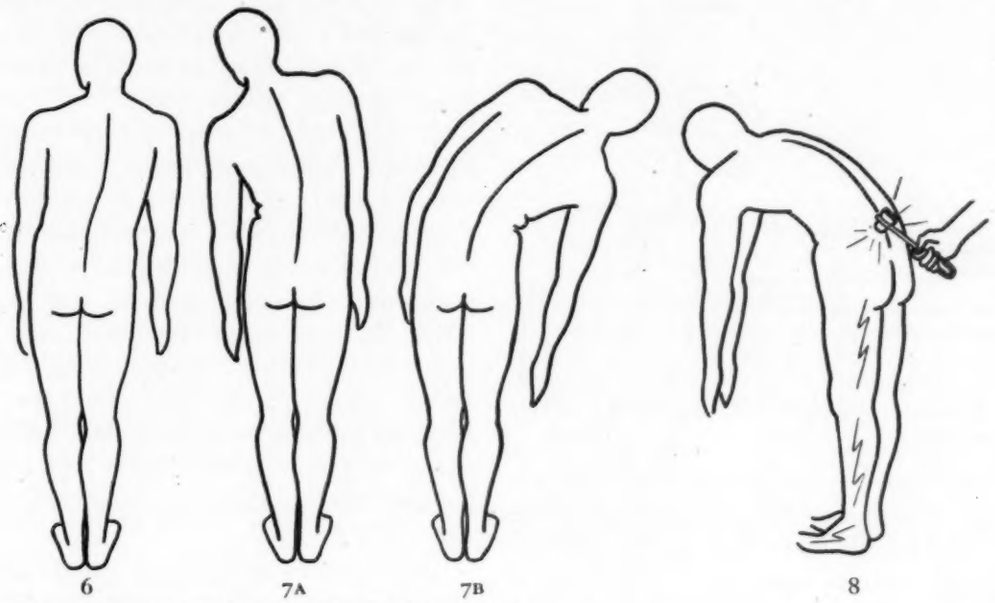
There may be one or more attacks of backache before the onset of sciatic pain. However, when trauma produces gross injury to the intervertebral disc, leg pain may occur simultaneously with low back disability. Once leg pain has started it usually follows the entire course of the sciatic nerve. The points of maximum intensity are the gluteal region, the posterior

thigh and the calf of the leg. Usually the sciatic pain remains unilateral, but occasionally it may be bilateral, and in a few cases the pain will shift from one leg to the other during the same or successive episode.

Some patients will voluntarily report that certain parts of their leg are numb and that there is a feeling of "needles and pins" in certain skin segments of the leg and foot.

Partial or complete remission of symptoms is the rule when the history covers a period of several months or years. Recurrent attacks with freedom from pain between the attacks is the usual history.

Many patients report relief of symptoms by manipulative therapy. Others apparently are made worse by such procedures and obtain no relief until they have remained in bed for a few days.



6 7A 7B 8

Fig. 6. Typical list from lumbar disc lesion (left).
Fig. 7. A, restricted lateral bending toward painful side; B, unrestricted lateral bending toward normal side.
Fig. 8. Production of radicular pain by deep percussion.

EXAMINATION

The following tests have been selected as being the most useful ones in the diagnosis and the localization of a ruptured intervertebral disc. These tests must be made carefully and the results recorded unequivocally. When doubtful the test should be considered negative.

1. Most patients with a ruptured intervertebral disc have a straight lumbar spine with obliteration or even reversal of the normal lumbar lordosis. Most of them, in addition, show listing away from the side of the sciatic pain. (Fig. 6.) In almost every instance the pelvis on the affected side is higher than on the unaffected side. Movements of the lumbar spine are usually limited on flexion. The erector spinae muscles may be spastic and the spasm usually is greater on the side opposite the lesion or painful leg. Lateral bending to the side of the lesion is usually more painful than when bending away from the painful side. (Fig. 7.) Occasionally, one encounters a patient with alternating scoliosis; in other words, he can change his list from one side to the other simply by bending lateralward or forward.

2. Another test which we have found to be very valuable is extension of the lumbar spine combined with lateral bending toward the painful side. Aggravation of the back and leg pain with numbness and tingling

into the distal portion of the dermatome during this maneuver is considered pathognomonic of a ruptured disc. The test is performed with the patient erect. It is important that the knees are straight and that both heels are on the floor. The

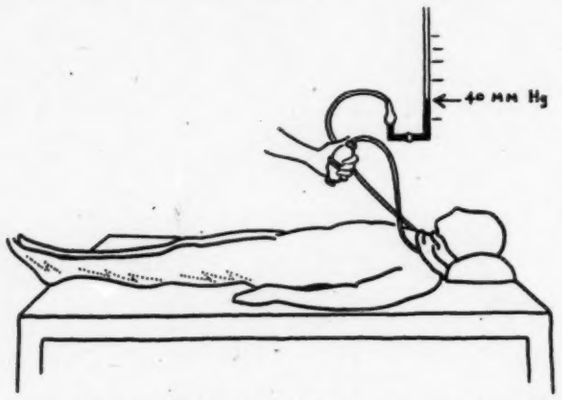


Fig. 9. Recommended method for performing jugular compression test.

examiner then grasps the patient's shoulders and slowly extends the lumbar spine and at the same time bends the patient toward the painful side.

3. *Radiating percussion pain* is also diagnostic. With the patient bent forward to the point of painful radiation into the leg a light percussion stroke over the spinous processes may accurately localize the tender point. A deeper percussion stroke to the side of the midline at this level may cause radiating pain into either the hip or down the back of the thigh or even into the calf. Radiating percussion pains thus produced are caused by waves of force transmitted through the ligamentum flavum to the affected nerve root at the site of the lesion. When positive this sign is pathognomonic of a ruptured intervertebral disc. (Fig. 8.) One may sometimes differentiate between L4 and L5 lesions by producing the radiating leg pain with the blow at one space and not at the other.

4. *The jugular compression test* is performed by impeding the venous return from the internal and external jugular veins with either digital compression or with compression by the cuff of a sphygmomanometer placed around the patient's neck. By increasing the intracranial pressure and consequently increasing the intraspinal pressure, the radicular pain in the affected leg may be aggravated. In fact, the entire pattern of the pain and even the sensory disturbances may be reproduced. (Fig. 9.)

The test may be performed with the patient either supine or upright. The venous return should be impeded until the patient has a distinct feeling of fullness in his head. Many patients will state that they feel as though their eyes were "popping out." Shortly after this venous congestion has occurred, the patient whose test is positive will state that the pain in the back is worse, that it radiates into the hip,

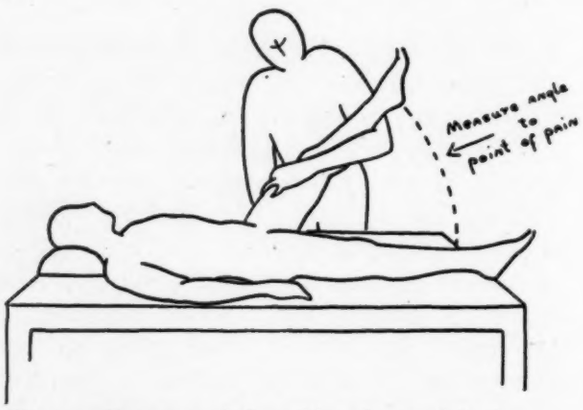


Fig. 10. Recommended position for straight leg-raising test.

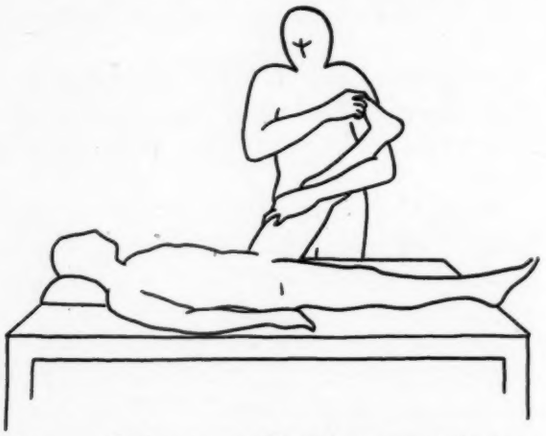


Fig. 11. Lasègue's modification of straight leg-raising test.

then down the back of the thigh into the calf. He may state voluntarily that one or the other of the toes feels numb or tingles. Often by careful observation during this test the localization of the lesion may be made with accuracy. The jugular compression test when positive is pathognomonic of an intraspinal lesion involving one of the nerve roots at the lower end of the spinal canal. However, the test may be positive with cauda equina tumors as well as ruptured discs; but if other signs of ruptured disc are present and the jugular compression test is positive, it is unequivocal evidence favoring the diagnosis of this lesion.

5. *The straight leg raising test* is a valuable index of the degree of sciatic pain. (Fig. 10.) The angle at which the pain appears may also be used as a reliable index for judging the progress of the case. The Lasègue's test is a useful refinement in equivocal cases. (Fig. 11.) These two tests are almost always positive with ruptured disc in the lower lumbar region.

6. *The sensory examination* is important in diagnosis, particularly if it conforms with the other clinical findings, especially the jugular compression test. Two forms of stimuli are sufficient to demonstrate gross sensory involvement—light touch with the fingertips and painful sensation with a pin prick. In mapping out areas of sensory loss it is desirable to compare the normal with the abnormal side. In many instances, the only sign of sensory disturbance will be the patient's statement that the skin of the involved area feels thicker than normal. In questionable cases, testing with heat or cold will demonstrate sensory loss when other tests are equivocal. Paresthesias may be described as peculiar sensations, electrical shocks spreading from the point of stimulation into the remainder of the involved dermatome. They are best reproduced by lightly stroking the skin with the fingertips. For sake of emphasis it will be restated that sensory loss on the top of the great toe indicates involvement of the fifth lumbar root at the fourth lumbar disc. Sensory loss on the lateral aspect of the foot involving particularly the lateral two or three toes indicates involvement of the first sacral root at the lumbosacral disc. Sensory loss on the medial aspect of the foot extending upward over the ankle indicates involvement of the fourth lumbar nerve and is characteristically found in lesions at the third lumbar interspace.

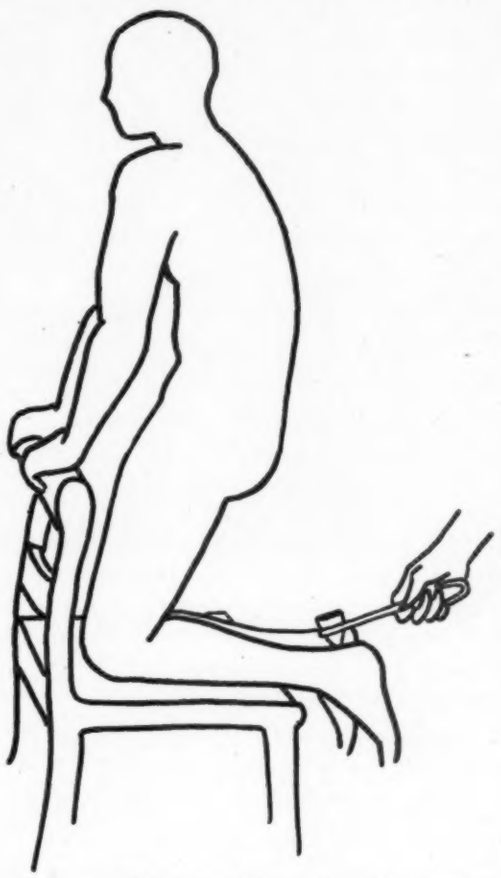


Fig. 12. Recommended position for testing Achilles reflexes.

7. *Reflex* changes are exceedingly valuable in diagnosis. With lesions at the third interspace involving the fourth lumbar nerve the knee jerk may be found diminished or absent. With lesions at the fourth interspace the only reflex change will be found at the posterior tibial tendon and that reflex is difficult to elicit consistently in normal subjects. Absence of the Achilles jerk is usually found with lesions at the lumbosacral disc. Since 98 per cent of ruptured discs occur at either

the fourth or the fifth lumbar interspace, the Achilles jerk is particularly important in differential diagnosis. It is a difficult reflex to elicit properly unless the examiner is thoroughly familiar with methods of producing relaxation and reinforcement of the tendon jerks. The position shown in Figure 12 is the ideal one for testing the Achilles reflex.

The reliability and accuracy of these signs have been tested upon thousands of patients by many observers. Most neurologists are in thorough agreement with their value and accuracy. If other physicians who are interested in the disc problem will study them and learn to evaluate them, the problem of the clinical diagnosis of ruptured disc will be greatly simplified.

SUMMARY OF CLINICAL SIGNS

Many ruptured lumbar intervertebral discs can be accurately localized on clinical findings alone. All cases have in common (1) low back disability and (2) pain in the distribution of the sciatic nerve. The characteristic differential points are as follows:

The Third Lumbar Interspace:

1. Tenderness to percussion lateral to the third lumbar spinous process.
2. Radiating percussion pain produced to the side of the third spinous process.
3. A positive jugular compression test with tingling appearing on the mesial aspect of the foot (the fourth lumbar dermatome).
4. The knee jerk diminished or absent; the ankle jerk normal.
5. Hypesthesia or paresthesias in the fourth lumbar dermatome.

The Fourth Lumbar Interspace:

1. Tenderness to percussion lateral to the fourth spinous process.
2. Radiating percussion pain produced to the side of the fourth spinous process.
3. Positive jugular compression test with tingling into the great toe (the fifth lumbar dermatome).
4. The knee jerk is normal and the ankle jerk is usually normal. Occasionally the ankle jerk is diminished. The posterior tibial reflex may be absent or diminished.
5. Hypesthesia or paresthesias in the fifth lumbar dermatome.

The Fifth Lumbar Interspace:

1. Tenderness to percussion lateral to the fifth spinous process.
2. Radiating percussion pain produced to the side of the fifth spinous process.
3. Positive jugular compression test with tingling appearing on the top of the foot and the lateral three toes (sensory disturbances are always absent in the great toe).
4. Diminished or absent ankle jerk; knee jerk normal.
5. Hypesthesia or paresthesias in the first sacral dermatome.

X-RAY AND LABORATORY AIDS IN DIAGNOSIS

Plain x-ray films of the spine are often helpful in diagnosis. The most constant and characteristic feature is straightening of the lumbar spine with absence or reversal of the normal lumbar lordosis.

Narrowing of one of the lumbar interspaces is presumptive evidence which may or may not be important. The fifth lumbar interspace is normally narrower than the other lumbar interspaces. Therefore, unless there is marked narrowing with spur formation about the joint such a finding is of little or no importance.

Localized proliferative changes about the diseased joint may or may not be of clinical significance. Not infrequently, the acute ruptured disc may be above or below the level of such a localized area of arthritis.

Congenital anomalies such as spondylolisthesis may or may not be of clinical significance. Such congenital lesions may indicate an unstable spine but in our experience does not give positive or negative evidence of a probable ruptured disc at the same level or above it. We are thoroughly convinced from repeated observations that spondylolisthesis *per se* does not cause sciatic pain. If it is of severe degree (grade 4), there may be widespread disturbance of the cauda equina function below the

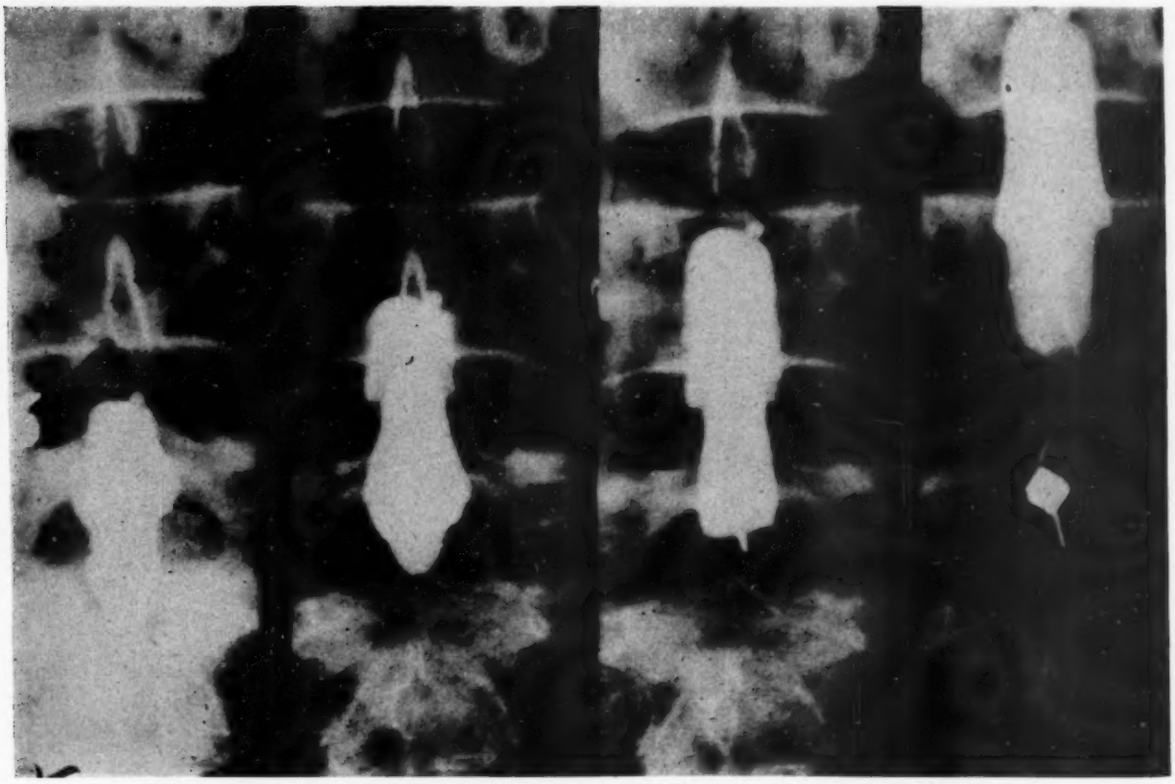


Fig. 13. Pantopaque myelogram showing normal column at L3, L4 and L5 interspaces.

level of the lesion due to obliteration of the spinal canal.

It is extremely important in studying plain films of the spine in patients with suspected ruptured intervertebral disc to observe carefully the sacroiliac joints. The Marie Strumpell type of spinal arthritis may produce symptoms strongly suggestive of ruptured intervertebral disc. Since this destructive type of arthritis usually shows up first in the sacroiliac joints, it is particularly important that the films be made in such a way that these joints may be accurately visualized.

MYELOGRAPHY

In this series of 350 verified ruptured discs, the diagnosis and localization were confirmed by myelography in 46 per cent of the cases. The remaining 54 per cent were accurately localized on clinical findings alone.

Pantopaque, an iodized oil of low viscosity, is the ideal medium for myelography. It is heavier than spinal fluid; therefore, it always seeks a dependent position in the subarachnoid space. By careful manipulation of the column under the fluoroscope it is possible to visualize accurately the subarachnoid space including the axillary pouches at any of the lumbar interspaces. If the x-ray machine is equipped with a spot film device, the operator can permanently record his fluoroscopic observations upon serial spot films. Once the examination is completed the material is usually aspirated without difficulty, leaving no trace of iodized oil in the subarachnoid space. Should a few drops of the oil be inadvertently left, it is slowly absorbed over a period of from three to four months and apparently produces no clinical symptoms of consequence. Pantopaque myelography is no more accurate than the skill with which it is done. In the hands of experts we are convinced that it is fully as accurate as the diagnosis of peptic ulcer with barium. However, there are many tricks to the examination and before any one attempts it he should be thoroughly

FIG. 14. Pantopaque myelogram showing characteristic filling defect at L4 on the left.

FIG. 15. Pantopaque myelogram showing characteristic filling defect at L5 on the left.

instructed in the technic of injection and in making the necessary observations while the drug is in the spinal canal, as well as in the interpretation of the spot films. Otherwise the examination may be more confusing than helpful. (Figs. 13, 14 and 15.)

It is our practice to use myelography on those patients only who are severely handicapped by low back and sciatic pain without clear-cut neurologic findings. Furthermore, the patient must have answered all of the criteria for operation. It should be stressed that myelography is not necessary for diagnosis in a ruptured disc, but it is very useful in localizing the lesion.

LABORATORY EXAMINATIONS

Many observers have recommended studying the spinal fluid in cases of suspected ruptured disc, particularly to determine if the total protein is increased. This procedure in our experience has been a totally wasted effort. Only in those cases in which the presence of a spinal cord tumor enters into the differential diagnosis do we recommend diagnostic lumbar puncture before myelography.

The sedimentation rate is an important routine laboratory study. This may be the only positive information of an early Marie-Strumpell arthritis, whereas in an uncomplicated ruptured disc the sedimentation rate is never elevated.

SURGICAL CONSIDERATIONS

Selection of Cases. Patients in their initial episode of pain, i.e., low back and sciatic pain, should have prolonged conservative treatment, for a large proportion of them will get well without surgery. The duration of conservative treatment depends on many factors, not the least of which is the economic status of the patient. A wage earner with a family to support cannot be kept flat in bed for a month or two with no assurance that he will be back to his job at the end of that time. It may be advisable in such a patient if the objective findings are well defined to cut the period of conservative treatment to two weeks, particularly if at the end of that time the patient has shown no improvement. Ordinarily, the patients who get relief from conservative treatment show some improvement after a few days of bed rest.

Patients who have had recurrent episodes of low back and leg pain and in whom the diagnosis is unequivocal are candidates for surgery. However, it is exceedingly important never to subject one of these patients to surgery during a period of remission. They must be having severe pain at the time they enter the hospital for either surgery or myelography. Otherwise it may be difficult to localize the lesion accurately. It is our practice, no matter how typical the history, to have the patient wait for another acute bout of pain before entering the hospital for study or operation.

Low back pain alone is seldom, if ever, an indication for an operation upon a lumbar intervertebral disc. Patients with incapacitating low back disability are strictly an orthopedic problem, either for conservative treatment with braces, etc., or for spinal fusion.

Unilateral, or occasionally bilateral, sciatic pain which is refractory to conservative treatment is the only specific indication for lumbar disc surgery. Furthermore, low back and sciatic pain must be incapacitating. The mild discomfort that so many patients complain of is not an indication for either operation or myelography. Furthermore, most patients whom we consider eligible for surgery have positive neurologic signs; otherwise, they are referred to the orthopedist for conservative management.

CONSERVATIVE TREATMENT

This is an orthopedic problem and it will receive only a cursory discussion here. In our experience the most effective measures are: (1) Complete rest on a hard flat bed until the acute pain has subsided. (2) If the sciatic pain is severe, traction upon the affected leg may be beneficial. (3) A well fitting low back brace made to the patient's own measurements is helpful once the patient becomes ambulatory.

SURGICAL TREATMENT

This operation may be one of the most difficult to perform properly. It should not be attempted unless the surgeon has been thoroughly schooled in the technic of intraspinal surgery. An intimate knowledge of regional anatomy is mandatory. The operation should accomplish the removal of all the nucleus pulposus, loose pieces of posterior annulus fibrosus and cartilaginous plates. This objective is possible without destroying either the laminae or the articular facets.

It is most difficult to teach the novice to recognize the exact level at which he is operating. Almost always he will place the incision too high. To avoid this difficulty it is always advisable for the operator to identify the lowest interspace by palpating the sacrum through the open wound.

The incision should be made directly over the spinous processes centering on the level of the suspected ruptured disc. The incision should be sufficiently long to allow the operator to work in comfort. However, the experienced operator requires only a small incision. An incision 2 inches long is quite adequate. The muscles on the side of the pain are dissected from the spinous processes and a hand retractor (similar to the Hibbs retractor) is used to displace the muscles lateralward. This maneuver exposes the ligamentum flavum.

At the fourth interspace, and often at the fifth interspace, there is a ledge of lamina overhanging the ligamentum flavum. The operator should remember that the ligamentum flavum is attached to the undersurface of the lamina above and the uppersurface of the lamina below. When this overhanging ledge of lamina interferes with dissection of the ligamentum flavum, it should be removed with rongeurs up to the insertion of the ligament itself. The ligamentum flavum is then removed by sharp dissection, care being taken not to injure the spinal dura or the dura over the nerve root during the dissection. A small nasal punch is a valuable adjunct in dissecting the lateral margin of the ligamentum flavum. Once the ligament is removed the lesion should be easily demonstrated by palpation with an instrument for the imprisoned nerve root will be found pushed backward against the ligamentum flavum. The nerve root is gently dissected free from the bulging mass with pledgets of cotton and retracted gently toward the midline. The bulging mass of disc material is then removed piecemeal with a pituitary rongeur or a similar biting instrument. At this stage most operators experience difficulty with the plexus of veins on the anterior surface of the spinal canal. These are easily handled provided that certain precautions are taken during the dissection. In the first place, once the ligamentum flavum is removed the extradural fat should be gently pushed upward and downward from the nerve root, thus exposing this mass of veins. Then they should be gently pushed backward and packed away from the disc with pledgets of cotton attached to silk sutures to prevent their being lost in the wound. If a vein is accidentally ruptured during the process of packing, a piece of gelfoam saturated with thrombin may be placed on the bleeding surface and the whole mass packed upward and downward again with pledgets of cotton. This assures a perfectly dry field for the leisurely removal of the ruptured disc. Once the bulging mass has been removed the rongeurs are then placed within the disc proper and all loose fragments are excised piecemeal. During this maneuver it is exceedingly important that the operator identify the anterior longitudinal ligament or he may tear through the anterior portion of the disc and be in serious danger of damaging important structures at that level, notably the iliac arteries and veins. The dissection within the disc should be thorough but *most carefully done*. Several sharp curettes of varying shapes greatly facilitate the removal of these loose particles of nucleus pulposus and cartilaginous plates. When no further loose particles can be obtained with either the biting rongeurs or the sharp curettes, the operation is completed. We have routinely used small packs of gelfoam around the disc incision in order to mini-

mize the dangers of postoperative bleeding. We have also covered the dura mater where the ligamentum flavum has been removed with strips of gelfoam. Closure of the wound is then made in layers with sutures of black silk.

When one fails to find a frank bulging mass beneath the nerve root at the suspected level, the disc should be carefully exposed by traction on the dura toward the midline. Then the disc should be carefully palpated with a blunt instrument. Not infrequently one will demonstrate the bulging mass more toward the midline rather than in the usual lateral position; and furthermore, when such a bulging mass is palpated with a blunt instrument, it is found to be exceedingly soft and resilient. Often the blunt instrument will fall into the tear in the annulus at this site. This is positive evidence of a ruptured disc and, therefore, justifies complete radical removal of the disc.

On the other hand, one is confronted occasionally with a disc that shows no such weak places in the annulus fibrosus. The disc may feel somewhat soft to palpation but certainly one cannot demonstrate a weakened portion in it. In such circumstances we believe that the disc should *never* be opened; and if no frank rupture can be demonstrated above or below this location, the wound should be closed and the exploration listed as a negative one.

The choice of anesthesia is an individual matter. We prefer spinal procain—100 to 120 mg. This anesthetic lasts for approximately forty minutes and assures complete muscular relaxation. No supplementary inhalation or intravenous anesthetics are used.

SPINAL FUSION

The place of spinal fusion in the treatment of ruptured intervertebral discs is still a moot question. In some clinics primary spinal fusion, after the removal of a disc, is more or less a routine procedure. In other clinics it is used in only a small percentage of cases. The most usual indication for primary fusion is instability of the lower lumbar spine due to congenital anomalies. In some clinics the presence of arthritic spurs in the region of disc disorders is considered sufficient reason for a primary spinal fusion.

In our clinic we have never resorted to primary spinal fusion for ruptured intervertebral disc *under any circumstances*. We believe that combining the operation is not to the best interest of the patient because we believe that divided responsibility in a surgical case is always an undesirable factor. Our attitude is that if spinal fusion proves to be necessary in order to correct intractable low back pain it had better be done as a separate procedure. The disc operation, providing it is done skillfully, will not in any way interfere with the operative field of the orthopedic surgeon when it comes time for a spinal fusion.

In our experience relatively few patients have complained sufficiently of intractable low back pain to justify spinal fusion after disc removal. Since there is no way of predicting preoperatively which patients will have this pain, it seems undesirable to subject patients to a fusion until time has proved that simple disc removal is not enough to affect a cure. Furthermore, we have never been impressed with the fact that congenital anomalies in the lower lumbar spine play any part in disc symptomatology. We have repeatedly removed ruptured discs from the fourth interspace when there was in addition a well defined spondylolisthesis at L_5. Most of these patients recovered about as normally as the patients without congenital anomalies.

The presence of arthritic spurs has never seemed to us to be an indication for spinal fusion. It would seem probable that these spurs are nature's effort to fuse the vertebrae, and once this process is completed there is likely to be good bony fusion and therefore relief of symptoms.

AFTER-CARE

The operation for ruptured intervertebral disc is quite a simple one from the patient's point of view. There is practically no disturbance of weight-bearing surfaces other

than at the site of the ruptured disc. Since a large part of the disc is removed at the operation it always has seemed to us advisable to keep the patient in bed for two weeks. Our routine is to leave the patient completely recumbent for the first twelve days, allow him to sit on the edge of the bed on the thirteenth day and to walk, on the fourteenth day at which time he returns home. He is instructed to increase his activities gradually during the next two weeks, but under no circumstances is he to put any strain upon the back such as lifting or stooping during that period. At the end of four weeks he is permitted to increase his activities and if his work is sedentary in nature he can return to his desk. Laborers and those who indulge in occupations requiring bending and lifting operations are advised to remain away from their work for three months postoperatively and then gradually to resume it. No postoperative immobilization is recommended.

Certain patients will develop excruciating back pain during the postoperative period. It will often be associated with a marked list of the pelvis and with marked muscle spasm. They are usually comfortable in bed. This type of patient in our experience usually develops in the course of time a full-blown proliferative arthritis at the diseased joint, and once the joint is fused, usually within a matter of six months, the pain disappears spontaneously.

Patients suffering in this way can often be greatly relieved by a light body cast applied with the back in extension, usually by overhead traction. This should be worn for from four to six weeks at the end of which time it may be bivalved and discarded completely in most cases. A light plaster body cast has, in our experience, been much more satisfactory than a well fitting back brace.

RECURRENT SYMPTOMS

The disc lesion recurred at the original level and was verified by reoperation in 3.3 per cent of the patients studied in this series. No doubt some of the patients listed as unsatisfactory results have recurrence of their lesion which has not yet been verified by reoperation. It seems safe to say, therefore, that the incidence of recurrence, taking into consideration other possibilities connected with this follow-up study, would not exceed 10 per cent. These recurrences have been at varying intervals from a few weeks to two or three years after the initial operation.

The results of reoperation are excellent. We have had no instance in which a ruptured disc has recurred more than once.

RESULTS

Three hundred seventy-eight patients operated upon for ruptured lumbar intervertebral discs were selected for this follow-up study. The entire group constituted "private patients" operated upon personally by one of the authors. We found it impractical to use the clinic patients for this study because of inadequate follow-up facilities. In spite of the fact that the patients were classified as "private" they represent workers from every walk of life, including fifty compensation cases.

The series is a consecutive one. No patient diagnosed and operated upon for a lumbar intervertebral disc was excluded from the study.

The investigation divided itself logically into two parts: A *long term* and a *short term* follow-up study. The long term series, from January, 1939, to April, 1942, consisted of 260 cases. The short term series, from January, 1946, to March, 1947, consisted of 118 cases. The break in continuity was occasioned by the authors' period of military service. There was no significant difference in the results of the long and short term series.*

The same surgical criteria discussed in the text of this paper were, for the most part, followed in both series of patients.

In planning the questionnaire we considered it important to ask questions so simple that no one could misunderstand

* A detailed study of follow-up statistics will be reported in a later communication.

them. They were as follows: (1) Are you able to do your usual work (the same as before your illness requiring operation)? (2) Do you consider your operation to have been successful? (3) Do you continue to have leg pain? (4) Do you continue to have low back pain?

Of the 378 questionnaires sent out, 301 had been returned at the time this manuscript went to press. Three hundred sixty-two patients apparently received the follow-up letter; sixteen of them were returned unclaimed.

Of the total series (301 patients), 242 (80.4 per cent) stated that they were able to do the same work at which they were occupied prior to operation. Forty-five (14.9 per cent) stated that they were able to do their usual work part of the time but were handicapped by recurrent symptoms at other times. Fourteen patients (4.7 per cent) had to change their occupations because of persistent symptoms.

Of the total group (301 patients), 243 (80.8 per cent) considered their operation to have been successful. Twenty-nine (9.6 per cent) of them considered their operation partially successful, and the remainder, 29 (9.6 per cent) considered it unsuccessful. It is interesting to note some of the reasons given for considering the operation partially successful. One patient writes: "I can't take long automobile trips because my back gets tired." But for the most part patients who considered the operation partially successful were those who had definite episodes of recurrent low back or leg pain which temporarily incapacitated them.

One hundred forty-two patients (47.2 per cent) stated that they have no leg pain whatsoever. One hundred thirty-nine patients (46.2 per cent) continue to have an occasional mild pain in the leg but not severe enough to be incapacitating. Twenty patients (6.6 per cent) stated that they have aggravating leg pain constantly, in fact just as bad as before they were operated upon.

It is interesting to note that the question with respect to low back pain brought only 117 replies (38.9 per cent) claiming complete and lasting relief of this symptom. One hundred sixty-three patients (54.2 per cent) stated that they have occasional recurrences of low back disability although in most instances it is not incapacitating and is of short duration. Twenty-one patients (6.9 per cent) claim to be completely incapacitated for their normal activity by persistent low back disability.

We consider the two questions, (1) Are you able to do your usual work, and (2) Do you consider your operation to have been successful, to be of paramount importance in attempting to evaluate the results of lumbar disc surgery. As we have pointed out elsewhere in this paper no surgical effort can hope to restore a ruptured disc to a normal functional state. It is not surprising, therefore, that somewhat less than half of the patients have been completely free of all symptoms referable to their former disc disturbance since operation. That over 80 per cent of the entire group of patients are able to carry on their usual activities and are satisfied with their operation (for which in most instances they paid out of their own savings) speaks eloquently for the practical aspects of conservative disc surgery.

OPERATIVE MORTALITY

One patient of this series of 378 cases died on the eighth postoperative day; an operative mortality of .26 per cent. Death was due to fulminating influenzal pneumonia (confirmed by necropsy). Since the death occurred in February, 1939, the newer sulfa drugs and penicillin were not available for treatment.

It would seem logical to assume, therefore, that the operative risk for lumbar intervertebral disc surgery is no greater than that for non-complicated inguinal hernia.

SUMMARY

1. A thorough knowledge of the anatomic relations in the lower lumbar portion

of the spine and of the physiology of the intervertebral disc is essential for a clear understanding of the clinical features of this disorder. The pathologic changes which occur in a ruptured lumbar intervertebral disc are irreversible; no surgical effort can hope to restore its anatomic integrity.

2. We believe that the term "ruptured intervertebral disc with or without herniation of the nucleus pulposus" more nearly describes the anatomic and pathologic features of the disorder than any other. The term "hidden disc" seems to be an unfortunate one and in our experience usually represents a misnomer for a negative exploration. Multiple ruptured discs are rare in our experience (2 per cent).

3. Low back pain, uncomplicated by sciatic radiation, is seldom if ever an indication for operation upon a lumbar intervertebral disc.

4. Unilateral (occasionally bilateral) sciatica which is refractory to conservative treatment is the only specific indication for lumbar disc surgery.

5. Patients in their initial episode of pain should have prolonged conservative treatment. A large proportion of them will get well without surgery.

6. Most patients with ruptured lumbar intervertebral discs whose symptoms are severe enough to justify operation present unequivocal neurologic signs. Not only are these signs invaluable and accurate in diagnosis but they can be relied upon in over half of the cases for localization of the lesion.

7. No matter how classical the history of a ruptured intervertebral disc may be, a patient should never be operated upon in a remission. This is one of the most common causes for negative explorations.

8. Disc operations may be among the most difficult in surgery to perform properly. The operation should accomplish removal of all the nucleus pulposus, loose pieces of annulus fibrosus and cartilaginous plates.

9. A myelogram is not required for diagnosis; it should be made on clinical findings alone. However, myelography is extremely valuable in localizing the lesion when the neurologic signs are not clear-cut.

10. Primary spinal fusion together with simple removal of the ruptured intervertebral disc is not a desirable procedure. Those patients who continue to have incapacitating backache after simple disc surgery may require a secondary spinal fusion. A disc operation properly performed does not in any way interfere with subsequent fusion operations.

11. The follow-up results of 301 verified ruptured intervertebral discs treated surgically are presented.

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MÉNIÈRE'S DISEASE*

ITS SURGICAL TREATMENT BY DIVISION OF THE ACOUSTIC NERVE

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MÉNIÈRE'S disease, or Ménière's syndrome, is characterized by paroxysmal attacks of vertigo in the presence of tinnitus and impaired hearing which are usually unilateral. Ménière's classical description of the syndrome which bears his name was made in 1861;[1] and although the disease is fairly common, the etiology is still undetermined and ideas of treatment vary widely. The symptoms are of varying degree and the milder ones are either tolerable or possibly benefited by medical therapy. But the more severe and intractable form of the disease is best treated by division of the acoustic nerve.

This report deals with fifty consecutive cases in which the operation has been employed and the patients followed thereafter from one to eight years.

PATHOLOGY AND ETIOLOGY

Since Ménière's disease is of itself not fatal, there is a paucity of autopsy material for the study of the pathogenesis. Hallpike and Cairns[2] in 1938 made the first report on the histologic changes in the temporal bones of two patients who had had typical Ménière's disease. In each of these cases they described gross distention of the endolymphatic system together with degenerative changes in the sensory elements. Since then, there have appeared other reports[3–7] with comparable postmortem findings of alteration in the endolymphatic system. It is inferred from these reports that the dilatation of the endolymphatic system results from overproduction of endolymph, from its defective absorption, or a combination of both, and that the location of the lesion in Ménière's disease is in the inner ear. Others, particularly Dandy,[8] have maintained that the lesion is not located here but along the afferent pathway, probably in the acoustic nerve.

The sensory end organs in the inner ear are in the semicircular canals, the maculae of the utriculus and the sacculus, and in the organ of Corti of the cochlea. The vestibular portion of the acoustic nerve coming from the semicircular canals and the maculae, and the cochlear portion from the organ of Corti come together near the inner end of the internal auditory canal and for a few millimeters have a common sheath. Near the brain stem the trunk of the nerve divides again into vestibular and cochlear portions which pass separately to their respective nuclei in the medulla. Nowhere do the central pathways of these two nerves come in close proximity.

Crowe[9] concluded that a single lesion in the brain stem or cerebrum could not cause all the symptoms of Ménière's disease without involving other structures and that anatomically there are only two possible sites in which a single lesion could cause the symptoms: in the nerve trunk or in the inner ear. Of the two he favored the latter and pointed out that although Corti's organ and the vestibular end organs are widely separated, they have the endolymphatic system in common. Dandy,[10] on the other hand, who shared with Crowe the responsibility for the care of many patients with Ménière's disease, maintained that the lesion lies in the acoustic nerve and not in the end organs and that a lesion affecting both cochlear and semicircular canals would be highly unlikely. He considered Ménière's disease similar to trigeminal neuralgia, also a paroxysmal disease and believed to result from a lesion in or on

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the sensory root of the trigeminal nerve. Horrax[11] and Colemen and Lyerly[12] have concurred in this idea.

Although no gross or impressive microscopic alteration of the acoustic nerve has been found in autopsy specimens or in segments of the nerve resected at operation, the suggestion has been made[10,13] that pressure on the nerve or intrinsic changes in it may result from anomalous vessels, chiefly the anterior inferior cerebellar and auditory arteries. These arteries show great variability in their size, configuration and relation to the nerve but their rôle in the etiology of the disease is difficult to establish. That vertigo and tinnitus may arise from the acoustic nerve can be demonstrated by low voltage faradic stimulation of the exposed nerve in a patient awake. Also, that all three symptoms of the Ménière's syndrome can occur from invasion or compression of the nerve is demonstrated by such lesions as the acoustic nerve tumor and the aneurysm of the vertebral artery. Nevertheless, it is also true that such lesions are not found to produce the typical paroxysms of vertigo so characteristic of Ménière's syndrome.

Infectious or "toxic" neuritis of the acoustic nerve which has occasionally been suggested as possibly responsible for Ménière's syndrome seems unlikely in view of the chronic course of the symptoms and the absence of changes in the nerve. In addition, there is a notable lack of infectious or "toxic states" in patients with Ménière's disease.

A frequent argument given to refute the contention that the disease has its origin in the inner ear is that tinnitus may persist after total division of the nerve but I have also observed persistence of tinnitus after total removal of a tumor of the acoustic nerve and division of the nerve proximally. A plausible suggestion made by Atkinson[14] is that tinnitus may be considered a form of paresthesia which may persist even after the nerve is cut and regardless of where the original lesion lies. Others contend that since destruction of the inner ear by surgical means will abolish symptoms, or at least vertigo, this precludes the possibility of the lesion's existence in the nerve. But in dispute of this it may be recalled that in trigeminal neuralgia, a condition in which no one believes the lesion exists in the sensory end organs, the pain can be relieved by procainization of the superficial area in which the pain is felt. Thus none of the rationale for locating the lesion in Ménière's disease in either the end organs or the afferent pathway is entirely valid. Nor is reasoning clarified by the occasional encounter of a patient who has paroxysmal vertigo and tinnitus from an ear that is totally deaf and lacks evidence of response to vestibular tests, yet is relieved by intracranial division of the auditory nerve.

Empirical reasoning based on the beneficial results claimed for various forms of medical treatment has colored much of the discussion concerning the etiology of Ménière's disease. Atkinson,[15] in dealing with this aspect of the problem, has suggested that a basic fault, which can be influenced by a variety of medical treatments, could be an alteration in capillary permeability particularly in capillaries of the stria vascularis in the labyrinth. This explanation previously advanced by Brunner[16] and Portmann[17] is thought to have received support from the autopsy findings of dilatation of the endolymphatic spaces. Atkinson[15] suggests further that an increase in capillary permeability can be produced either by anoxia from a diminished blood supply which impairs the function of the capillary walls or by sensitization to some foreign protein or chemical. In either case the resulting alteration of the fluid content of the endolymphatic spaces might be the source of symptoms and of the reported changes in these spaces. While it seems to follow that periodic alterations in capillary permeability might coincide with paroxysms of vertigo and fluctuations in tinnitus, some organic changes must also take place to cause the loss of hearing and sometimes loss of response to vestibular tests.

In the fifty cases reviewed for this report, the disease occurred in thirty-three males and seventeen females, a ratio of two to one. The disease was exclusively or predominantly on the right side in twenty-six patients and on the left in twenty-four. It is largely a disease of middle life as is shown by the age of onset of first symptoms. In the first two decades there were no patients; in the third, eleven; in the fourth, sixteen; in the fifth, fourteen; in the sixth, six; and in the seventh, three. The youngest age at which symptoms began was twenty and the oldest sixty-two. These ages do not correspond to the ages which patients attained before coming to operation and while eleven developed their first symptoms in the third decade only three came to operation before the age of thirty, and there were five after the age of sixty.

Scrutiny of the patients' past histories failed to show with the possible exception of a few cases that the onset or progress of the disease might be significantly related to other diseases or to injuries. One dated the onset of unilateral deafness and tinnitus later followed by vertigo to a deafening explosion and a simultaneous head injury and concussion. Another dated the onset of vertigo to severe epidemic influenza (1918) and had comparatively frequent attacks of vertigo for the next twenty-two years. Still another experienced the simultaneous onset of vertigo, unilateral tinnitus and loss of hearing while recovering from an unidentified meningitis. Four had middle ear infections during childhood many years before the onset of Ménière's syndrome; one had a mastoidectomy on the side of Ménière's disease fifteen years before, and one elderly patient had a greatly sclerosed mastoid bone on the side of the disease. The general physical examinations and routine laboratory studies of all patients showed two with mild diabetes mellitus, three with mild hypertension, one with rheumatic heart disease and six with recurring paranasal sinusitis seemingly unrelated to Ménière's disease. There was a conspicuous absence of other diseases of the nervous system, tuberculosis, syphilis, blood dyscrasias, metabolic diseases, vasospastic and vascular occlusive diseases and of allergic disorders such as hives, asthma and intolerance of specific foods and drugs. There was, however, a comparatively high incidence of headache, some typically migrainous but mostly of a special variety which might be considered as a symptom directly or indirectly related to Méinère's syndrome.

SYMPTOMATOLOGY

The cardinal symptoms of Ménière's disease are paroxysms of vertigo, impaired hearing of the inner ear type which is usually unilateral and grows steadily worse, and tinnitus which is referred to the deaf ear. Aside from the tinnitus and deafness the patients usually feel perfectly well between the attacks of vertigo. In some patients all three symptoms have a simultaneous onset which makes early recognition of the disease relatively simple but this occurred in only 18 per cent of my cases. In other cases any one or a combination of two of the symptoms antedated the other symptom or symptoms by weeks, months or years. In 28 per cent of the cases unilateral tinnitus alone antedated the other symptoms, the longest interval being fifteen years. In 24 per cent vertigo alone antedated the other symptoms, the longest interval being ten years. In 16 per cent unilateral impairment of hearing alone antedated the other symptoms, the longest interval being twelve years before tinnitus, developed and, in the same patient, eighteen years before vertigo occurred. In 14 per cent various combinations of two simultaneously occurring symptoms antedated the appearance of the third symptom, the longest interval being six years.

From these data it is evident that incipient Ménière's disease often exists before the advent of the characteristic triad which makes recognition of the disease possible or reliable.

In eight cases or 16 per cent of this series there was bilateral tinnitus in addition to vertigo and hearing loss; and while it must

be recognized that bilateral Ménière's disease occurs in about this proportion, a special problem exists in these cases if unilateral nerve section is contemplated. Usually, there is some indication that one side is more involved by the disease than the other and this, from my experience, is the clue to the location of the offending side as far as the attacks of vertigo are concerned. In one case of bilateral tinnitus of equal degree the deafness was unilateral. In four cases of bilateral tinnitus the hearing loss was bilateral but greater on one side. In two cases of bilateral deafness of equal degree and bilateral tinnitus, the tinnitus was distinctly greater on one side. But in one case in which the bilateral tinnitus and bilateral deafness were each of equal degree the reliable clue to the location of the offending side, as far as vertigo is concerned, appears to have been a "dead" labyrinth on that side. These conclusions seem justifiable in view of the successful outcome of unilateral acoustic nerve section in each case. There were no cases in which bilateral deafness and unilateral tinnitus occurred.

Vertigo. Vertigo is the most striking and most distressing feature of Ménière's syndrome and the symptom for which patients chiefly seek relief. The sensation is one in which surrounding objects seem to whirl or jump in any direction within a horizontal or vertical plane. However, the direction and plane are of no value in determining from which side the trouble arises. As Crowe[9] has recalled, the discrepancy in the direction of the vertigo can be reproduced in normal persons by alternating the temperature of the water in the caloric test or alternating the direction of the rotation in the Barany tests. The vertigo characteristically recurs in attacks that are usually sudden, sometimes so sudden that the patient is thrown to the floor or is rapidly propelled off balance. Three of the patients of this series had sustained fractured bones in falls during attacks. The attacks last from a few seconds to many hours and occasionally for several days; the longest attack in a patient's experience is likely to be his first one. During an attack the patient must support himself or lie down; and though some find benefit from lying motionless on one side or the other, unrelated to the side of the disease, the attack for the most part seems to run its course unaffected by position. Two patients reported that they could suspend the vertigo as long as they could forcibly deviate the eyes in a special direction. In one of these observed during an attack, deviation of the eyes away from the direction of the nystagmus was said to suspend the vertigo but the patient was unable to hold the eyes in deviation for more than a minute at a time.

The intervals between attacks of vertigo vary in different patients and in the same patient. It is the unpredictability as well as increasing frequency of the attacks which bring most patients to consider operation. Spontaneous recovery from attacks undoubtedly does occur sometimes and similar to the attitude that patients with trigeminal neuralgia often take, there is the hope that each attack may be the last. In this series eight patients had remissions from vertigo for a year or more and one for fifteen years, but after these long remissions the frequency of the attacks steadily increased. In the early part of the disease the patients are usually free of any sense of vertigo or of sensations of imbalance between attacks though some in the later period of the disease have a more or less constant feeling of giddiness, unsteadiness or swaying which may be accentuated by movements of the head or sudden changes in position of the body. But patients who complain only of dizziness, swooning, giddiness, unsteadiness or uncertain sensations in the head without definite episodes of vertigo do not have Ménière's disease and this feature alone can be relied upon to differentiate these commoner complaints from those of Ménière's disease.

While in the majority of cases there are no aura preceding attacks of vertigo, in six patients (12 per cent) there were premoni-

tory symptoms present from a few minutes to several hours which included increased tinnitus, increased impairment in hearing, a feeling of fullness or aching in the head and ear on the side of the lesion, flushed feeling in the face on the side of the lesion, numbness in the hands and throat and faintness.

In all but two patients vomiting occurred with vertigo though in the majority it accompanied only the more severe attacks of vertigo. It was not preceded by nausea in some and a few denied ever experiencing nausea even in prolonged attacks of vertigo with vomiting. Some patients were so impressed by the vomiting that they tended to minimize the vertigo and attributed the entire episode to "upset stomach." Other symptoms which are commonly recognized as accompanying nausea and vomiting from any cause and need not be considered as peculiar to a Ménière's attack included, chilliness, sweating, headache, palpitation, borborygmus and diarrhea.

Tinnitus. Tinnitus of Ménière's disease is not any different in its characteristics from that which occurs from many other conditions, whether in the peripheral mechanism or central pathway for hearing. It was present in all cases of this series and bilateral in eight. The term "tinnitus" strictly speaking means a ringing, but in common usage it includes sounds variously described as hissing, rushing, buzzing, sizzling, whistling, swishing, screaming, roaring, beating, drumming, vibrating or explosive. There is great variation in quality, pitch, intensity and in frequency of alternating sound even in the same patient and the purely ringing sound may be described as humming, singing, tinkling or gong-like. A sailor was particularly distressed because his tinnitus was often indistinguishable from a bell buoy and a typist could not always distinguish hers from the warning bell on her typewriter.

There are two modes of onset of tinnitus in Ménière's disease. One is abrupt and loud and usually persistent, and is frequently attended from the start by impaired hearing. The other is gradual and more or less intermittent but steadily increasing in pitch and frequency with the passage of time. In about half the patients studied there was some increase in the tinnitus noticed before, during or after attacks of vertigo.

While tinnitus constitutes one of the complaints in patients with Ménière's disease, it is not the predominant complaint. In my experience most patients confronted with the decision of an operation intended to relieve vertigo but not promised to relieve their tinnitus show comparatively little concern over the prospect of continuation of their tinnitus if vertigo is relieved. In patients with or without Ménière's syndrome who are totally deaf or in a state of anxiety tinnitus may become an obsession.

Deafness. Loss of hearing in Ménière's disease has all the characteristics of deafness of a cochlear or acoustic nerve lesion. Both air and bone conduction are impaired and although the audiometric curves are variable, in most instances all tones are more or less affected. The hearing loss is not usually total but in most patients that come to operation the hearing remaining on the side from which attacks arise is of little practical value. Like tinnitus, the deafness may have an abrupt or a gradual onset with an insidious progression that makes it difficult for the patient to be certain of its beginning and sometimes unaware of its presence. After it progresses to a point beyond useful hearing and short of total deafness it usually remains stationary. Temporary increase in deafness is often evident at the time of an attack of vertigo. Only three patients had relatively normal hearing by audiometric tests and none of these had a complaint of deafness. In the majority, the loss was between 35 and 60 per cent in the diseased ear which is in a range below useful hearing. Seven patients had significant loss of hearing in both ears.

Other Symptoms and Signs. The occurrence of some form of headache in many of the patients with Ménière's disease has led a few to suggest that the two condi-

tions may have a common etiologic vascular mechanism. In this series, six of the patients had been subject to recurrent headaches prior to the onset of the first symptom of Ménière's disease and this is about the percentage in which headaches occur in the general populace. The incidence of headache after the advent of Ménière's syndrome was considerably higher (about 50 per cent) but includes a variety of headaches. In many it was bilateral or generalized and not impressively related in time of occurrence to the attacks of vertigo and considered to be probably a manifestation of an anxiety state based on a justifiable fear of unpredictable attacks of vertigo. There were other patients (18 per cent), however, with symptoms about the head more suggestive of some direct relationship with Ménière's disease. These symptoms included a sense of fullness, pressure or aching and even sharp pain in the ear, about the ear or some part of the head on the side of the disease which preceded, accompanied or followed an attack and sometimes occurred between attacks. A few mentioned a sense of flushing and even aching in one side of the face prior to an attack. Three described a feeling of a "dead cheek" occurring on the side of the tinnitus and deafness, particularly with attacks of vertigo and two of these appeared to have demonstrable sensory impairment in the side of the face including diminished corneal sensation. In these latter cases the numbness disappeared after division of the acoustic nerve.

Visual disturbances including blurring, diplopia and hallucinations of flashes, rings or scotomas occurring during and after an attack of vertigo were reported by nearly half the patients but with the exception of the hallucinations the symptoms might readily be explained on the basis of the nystagmus which accompanies the attacks.

Loss of consciousness occurring for short periods with the onset of more severe attacks of vertigo was reported by four patients. In each of these there has been no recurrence of loss of consciousness following relief of the attacks of vertigo by operation, which suggests that the syncope may have resulted from fall in blood pressure that came with the attacks of vertigo.

DIAGNOSIS

The diagnosis of Ménière's disease is made almost wholly on the history and should not be difficult if attention is given to the description given by the patient and if the triad of symptoms is present, namely, attacks of vertigo, tinnitus and deafness. While vertigo alone may be a manifestation of the disease, from a surgical standpoint the diagnosis cannot be made until tinnitus or deafness or both develop. In this series every patient had all three symptoms. Nystagmus undoubtedly occurs with every attack of vertigo (although I have observed few attacks) and in some it persists for a while after an attack but even when its direction is observed no definite clue can be gained as to the side of the lesion. McKenzie[18] quotes the case of a physician who observed nystagmus on himself on frequent occasions during attacks and found variation in different attacks.

The vestibular tests are not always reliable as an index of function for the results seem to vary in some patients, but the caloric test was performed one or more times in thirty-eight patients of this series. The method suggested by Atkinson[19] was used routinely but in those in whom this test produced no response more prolonged irrigation of the ear was employed. The results showed that of the patients tested the responses were normal in 35 per cent and absent on one side in 12 per cent; by comparison of the two sides there was a diminution of vestibular response in 33 per cent and an increase in 20 per cent on the side of the lesion. These figures show that the alteration in caloric response is of little value in the diagnosis yet there was one case in the series in which the test seemed to prove useful. This was the case already referred to in which tinnitus and

deafness were equal bilaterally and the absence of caloric response gave the clue to the side on which successful operation was performed.

In differential diagnosis there are few other diseases to be confused with the complete syndrome of Ménière's disease. The only diseases of importance are the cerebello-pontile angle tumors and usually there are other signs that make the diagnosis unmistakable. Dandy[10] reported encountering three unsuspected tumors when operating for Ménière's disease but no difficulties should arise from such a mistake since the operative exposure is the same for both lesions.

MEDICAL TREATMENT

A variety of medical treatments for Ménière's disease has been employed over the years, and anyone attempting to evaluate them quickly finds himself at a loss. The subject has been reviewed several times in recent years.[20,21,22] All of the current medical treatments are claimed to be predicated on clinical experiments made by their originators and the usual criteria taken for success is relief of vertigo though improvement in tinnitus and occasionally in hearing is also reported. Good results are reported to occur in from 62 to 96 per cent of patients;[22] and while the figures seem suspiciously high, undoubtedly some of the claims for improvement must be justified. Whatever the figures are it is only fair to conclude in our present state of knowledge of the disease that most patients should be given a trial period of treatment before resorting to surgery.

One method of treatment restricts fluid intake (Mygind and Dedering)[23] and another (Furstenberg)[24] attempts to accomplish the same end by dehydration with large doses of ammonium chloride; others seek to alter the electrolytic balance (Talbott and Brown)[25] while still others attempt to produce vasodilation with histamine (Horton and his collaborators)[26,27] and nicotinic acid (Atkinson)[28] or to produce desensitization by histamine (Atkinson).[29] Perhaps each method has some advantage in particular cases.

In this series 70 per cent of the patients had some form of medical treatment and over half of them had intensive treatment before coming to operation.

SELECTION OF CASES FOR SURGERY

The indication for operation in patients with Ménière's disease is based on the frequency and severity of the attacks of vertigo and the degree of disability produced. The majority of patients with Ménière's disease do not require surgical treatment since (1) the symptoms are mild or infrequent; (2) sedatives or some more specific form of medical treatment may be useful; (3) the attacks may in time subside spontaneously or (4) some co-existing disease may make the risk of an operation unjustifiable.

No patient should be considered for operation unless he has had the disease long enough to make it possible to rule out labyrinthitis or any other condition which may be confused with Ménière's disease in the early stages. In this series the shortest interval between the onset of vertigo and operation was ten months, the longest twenty years, and the average interval was four and a half years.

In addition to vertigo a lateralizing symptom or sign, namely, unilateral tinnitus or deafness must exist. In case of bilaterality of the tinnitus or deafness there must be a predominance on one side to indicate the side on which the operation is to be performed. In case the operation is to be performed on the only side in which useful hearing remains, consideration must be given to the possibility of additional loss of hearing in the better ear from the operation.

But in the last analysis decision for operation should be entertained only when the patient's comfort or ability to carry on his occupation is compromised by his attacks of vertigo. Relief of tinnitus is not a predictable outcome of the operation and hearing is not improved. Whereas the

operation of division of the auditory nerve intracranially should carry a low mortality (less than 1 per cent), it is none the less a major operation and carries a potential risk to life.

SURGICAL TREATMENT

Historically it is interesting to note that in 1874 Charcot[30] in demonstrating cases of Ménière's disease observed that when deafness became complete the attacks stopped spontaneously and he was prompted to suggest that division of the acoustic nerve might be of benefit. Ballance,[31] in 1894, also suggested division of the eighth cranial nerve for the relief of vertigo. Krause was credited by Frazier[32] with having sectioned the eighth nerve in 1905 for the relief of tinnitus. In 1908 both Frazier[32] and Ballance[33] sectioned the nerve, one for the relief of dizziness and the other for "painful tinnitus." Frazier[34] again performed the operation in 1913 for tinnitus. None of these early operations was attended by striking success, apparently because the cases were not ideally suited and the results did not make enough impression to bring the operation into general use.

The operation of intracranial division of the acoustic nerve for Ménière's disease has been popularized by Dandy[8] who made his first report of successful results following total section of the nerve in 1928. There have followed in the succeeding years equally enthusiastic reports on the operation by Coleman and Lyerly,[12] Cairns and Brain,[35] Olivecrona,[36] Munro[37] and Adson.[20] McKenze,[38] in 1931, began partial section of the nerve designed to interrupt the vestibular fibers but spare the cochlear fibers, and Dandy[39] reported on a similar procedure in 1930, in which he suggested section of five-eighths of the cephalad portion of the acoustic nerve. Dandy,[40] in 1935, reported the successful treatment of bilateral Ménière's syndrome and recommended division of the vestibular portion of each nerve. Although no other neurosurgical clinic has had occasion to perform division of the acoustic nerve for Ménière's disease in numbers even remotely approaching those reported by Dandy (401 cases in 1941[10] and over 600 cases in 1945[41]), the operation is widely accepted by neurosurgeons here and abroad as being well suited to selected cases.

Other less commonly performed surgical forms of treatment of Ménière's disease have been directed toward the end organs of the inner ear. Portmann's[42] operation on the endolymphatic sac has relieved some patients (Waltner)[43] but the relief has often been only temporary. In England, a commonly used operation has been alcohol injection into the labyrinth (Wright)[44] either through the drum and oval window or through a trephine opening in the horizontal canal. This procedure causes destruction of both the cochlea and vestibule and sometimes causes facial paralysis. Putnam[45] reported two cases in which he coagulated the vestibule through an opening into the superior semicircular canal by way of a transtemporal approach but the approach seems unnecessarily difficult. Ablation of the membranous organ of the horizontal canal or vestibule by mechanical destruction or alcohol injected through the mastoic route has been reported by Mollison[46] and Cawthorne.[47] These procedures also destroy both cochlear and vestibular function. The procedure reported by Day[48] employs the same mastoid approach but seeks to destroy by coagulation only the vestibule and preserve hearing. By comparison with these operations on the inner ear, it appears that intracranial total or subtotal division of the acoustic nerve is more direct and selective and, as a result, more reliable in its results.

Technic of Intracranial Division of the Acoustic Nerve. In all but three cases of this series the anesthesia employed was ether vapor administered through an endotracheal tube; in the others local procain infiltration of the scalp was used. The horizontal cerebellar position was used routinely although the upright position recommended by Adson[20] may have advantages. The in-

cision in the scalp was unilateral and semicircular with downward reflection of a soft tissue flap sufficient to give a good exposure of the occipital bone on the side of the lesion. Others recommend a straight vertical lateral incision for it can be made with slightly more speed and the occipital nerve may be preserved, but in case difficulties arise the exposure permitted by this incision may be inadequate. A window is made in the occipital bone which borders on the mastoid cells and is of sufficient size to expose the lateral half of the lateral venous sinus. If mastoid cells are accidentally or deliberately opened, they may be sealed with bone wax and additional safety provided by administering penicillin postoperatively. The dura is opened and fluid released from the cisterna magna through a small puncture in the arachnoid. This permits easier retraction of the cerebellum in exposing the region of the cerebello-pontile angle. The release of additional fluid from the lateral cistern in the angle facilitates additional retraction of the cerebellum for exposure of the acoustic nerve. The retractor should be as close and as nearly parallel as possible to the tentorium. Any veins which pass from the cerebellum to the tentorium that may be torn by the retraction should be coagulated. Small vessels, either arteries or veins, about the nerve should be gently brushed aside or coagulated and divided if necessary in order to obtain clear exposure of the nerve. Occasionally, the facial nerve can be partly seen beneath the cephalad border of the acoustic nerve but usually it is desirable to separate the two gently with a nerve hook. Separation of the two nerves is not always easy but is necessary in order to avoid damage to the facial nerve and facial paralysis. While the hook lifts the acoustic nerve, the nerve can be partly or wholly divided with a knife or the hook may be touched with the cutting current of the electrosurgical unit. In partial division of the nerve when it is desired to interrupt only the vestibular fibers and preserve the major portion of the cochlear fibers, the anterior or cephalad half, or slightly more, of the nerve is cut. In the closure of the wound it is desirable to approximate the dura accurately and minimize leakage of cerebrospinal fluid by sealing off the line of closure with gelatin foam strips. The soft tissue flap is restored to its position and accurately approximated in layers with interrupted silk sutures. Only a covering dressing for the wound is necessary though I have found some advantage in maintaining moderate pressure over the area for a day or so by sponge rubber held in position with a gauze roll encircling the head. This serves to minimize the collection of fluid and blood between the dura and muscle. When properly done, the operation carries very little risk and the patient can usually get out of bed on the second or third day.

Results of Operation. Unilateral operations were performed in all patients of this series even in those who had bilateral symptoms of one kind or another and thus far there has been no need for considering division also of the nerve on the opposite side. One patient of sixty-two years died from a cerebral hemorrhage which occurred on the fifth postoperative day and was verified at autopsy. One patient developed a complete facial paralysis which, however, recovered after several months; it resulted apparently from the manipulation necessitated in the control of bleeding from a torn petrosal vein. Three patients developed very slight facial weakness which disappeared within two weeks after operation.

In twenty patients partial division of the nerve was performed and in thirty the entire nerve was divided. All but two were completely relieved of vertigo. The two that were unrelieved had partial nerve sections and by postoperative caloric tests were found to have vestibular fibers remaining. In one of these a total division of the nerve was performed three months later with complete relief of vertigo. The other patient found that her attacks of vertigo, though persistent now for seven

years postoperatively, have been significantly diminished in frequency and degree and she has been unwilling to submit to a second operation. As a result of this experience it is my conclusion that failure of the operation to relieve vertigo in properly chosen cases of Ménière's disease (at least in unilateral disease) results from incomplete division of the vestibular fibers. For this reason total division of the nerve is preferable unless there is some special reason for preserving the residual hearing in the diseased ear.

In the early postoperative period there is usually a complaint of more or less dizziness which I have rarely found to be described as vertigo. Associated with this there is usually nystagmus of moderate degree when the eyes are deviated away from the side of the operation. Blurred vision difficulty in focusing and occasionally diplopia are also frequent complaints in the early stages after operation. But all of these subside steadily and are usually gone or are minimal within a few days to several weeks. Only a few patients will mention these disturbances for longer periods of time and none in this series was incapacitated by them after the initial period of recovery from operation. In all, the Rhomberg test was negative. Patients who have a dead labyrinth prior to operation are likely to be troubled very little or not at all by these symptoms. The majority of patients resumed their normal activities in from three weeks to three months after operation.

Tinnitus is unfortunately not relieved in all patients but may be improved and is rarely worse. In those in whom the tinnitus is improved but not eliminated there is less intensity and lowering of the pitch. Greater improvement in tinnitus followed total division of the nerve than followed the partial division. In the patients having total nerve division tinnitus was abolished in 30 per cent, definitely improved in 40 per cent and unimproved in 30 per cent. In patients having partial nerve division tinnitus was abolished in only 15 per cent, improved in 25 per cent and unimproved in 60 per cent. This discrepancy in the effect of the two types of operation on tinnitus is perhaps an additional reason for favoring total division of the nerve.

Hearing in those patients subjected to partial division of the nerve was not found after operation to be improved by audiometric tests though some believed they could hear better particularly if tinnitus was diminished or eliminated. But some of the patients with total unilateral nerve section also expressed the belief that their ability to hear was improved and only a few believed their useful hearing to be worse. It is a common belief that residual hearing following partial section of the nerve is progressively lost thereafter but this has been actually observed by symptom and audiometric tests in only one patient of this series.

SUMMARY AND CONCLUSIONS

Patients with Ménière's disease may be benefited by medical treatment; but if the treatment is ineffective, they should be advised to choose operation. Other patients whose occupation may make the risk of an attack of vertigo hazardous or whose economic status does not permit the loss of time should be similarly advised.

Of the several forms of surgical treatment intracranial division of the acoustic nerve gives the best results.

A review of fifty cases in which total or partial intracranial division of the acoustic nerve was performed shows that in a follow-up period of one to eight years all but one was relieved of vertigo.

Failure to relieve vertigo in unilateral Ménière's disease is due to incomplete division of the vestibular fibers.

Except when it is especially desirable to preserve hearing total division of the acoustic nerve has an advantage over partial division in that it is easier to perform, all vestibular fibers are unquestionably cut and there is a greater likelihood of obtaining relief from tinnitus as well as vertigo. Usually patients with hearing

already impaired by Ménière's disease do not find total deafness in the diseased ear an increased handicap.

The morbidity and mortality of the operation are not formidable.

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Pontine tumors produce extensive cranial-nerve disturbance, usually early and bilateral. Pyramidal-tract involvement is greater than in other posterior fossa tumors.

ABSCESS OF THE BRAIN*

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THE incidence of brain abscess formation in recent years has decreased tremendously. The disease, however, is still common enough to command a continued interest in this subject. The literature over the past fifty years is voluminous and a review of these articles leaves little or nothing new to be added. A few of these outstanding publications may be classed as surgical milestones in the treatment of brain abscesses and as such justify calling attention to them again.

It is believed that the same therapeutic agents that have been responsible for the total reduction in the number of brain abscesses have also served to alter some features of the symptomatology as previously recognized in these lesions. With a lull in the acute symptoms, a false security, thwarted with serious potentialities, has resulted. In the past five years, newer surgical ventures fortified with newer therapeutic agents offer some advantages and more assurance of a cure over previous methods of treatment. The experiences set forth in this communication are based on a study of fifty-six patients with abscesses located within the cerebral or cerebellar tissues. An attempt to depict the symptomatology and evaluation of the various types of treatments instituted in these fifty-six personal cases will be undertaken after a brief reference to the more outstanding surgical contributions on this subject. (Table 1.)

It would be short of sacrilegious to develop a monograph on the subject of brain abscesses without tribute to the work of Macewen.[1] In his selected cases published in 1893, he charts ninety-four patients with infective intracranial lesions in which seventy-four were operated upon; sixty-three were "cured" and thirty-one died. Of twenty-five cases of intracranial abscess, nineteen were operated upon and eighteen were "cured." One death occurred. This contribution set the score which neurosurgeons have since attempted to equal. Dowman[2] advocated in 1923 the production of adhesions between the cortex and the dura before the drainage of an underlying abscess. In 1924 King[3] advocated cortical uncapping to allow abscess extrusion. Dandy[4] in 1926 recommended simple aspiration as a method of treating chronic abscess formations and as late as 1945[5] was still inclined to "do the least possible." In 1928 Sargent[6] reported five encapsulated abscesses removed *in toto*. Coleman[6] in 1929 reported twelve recoveries in a total of fourteen patients treated by drainage, "using the eye-end of a small rubber catheter." In 1937 Vincent and Askenasy[7] reported their successes in total extirpations, resorting in some to osteoplastic decompression without opening the dura. Tapping the abscess was sometimes necessary to age the lesion for later enucleation. The abscesses were removed intact. Grant[8]

TABLE 1
FIFTY-SIX PATIENTS WITH ABSCESS OF THE BRAIN—FOURTY-SEVEN SURGICALLY TREATED WITH EIGHTEEN DEATHS

	No. of Cases	Deaths	Living
Autopsy (No Surgery)	9	9	0
Aspiration (Only)	12	5	7
Aspiration (Later Drainage)	2	2	0
Drainage (Only)	19	6	13
Decompression and Aspiration	3	2	1
Resection	9	1	8
Decompression (Only)	2	2	0

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in 1941, listing a bibliography of 207 references, so thoroughly covered the subject of brain abscesses in his collective review as to leave nothing but to quote his closing opinion: "Selected case series show a relatively satisfactory mortality rate, if neurosurgeons or otological consideration were taken of every case admitted to a clinic in which a final diagnosis of brain abscess was made by operation or autopsy, whether the abscess was acute or chronic, adjacent or metastatic, solitary or multiple, and regardless of complications or the patient's condition or admission, the average mortality rate would be about 40 per cent." In 1945 the author[9] reported five cases of total extirpation of abscess lesions, all of which had been totally removed either piece-meal or by aspiration at the time of craniotomy. One might offer the conclusion from the literature on this subject that the problem of chronic abscess treatment should offer no greater surgical mortality than that of surgery of intrinsic cerebral tumors and totally removed abscesses, the morbidity should be no greater than that following the total removal of a benign tumor similarly located.

SYMPTOMS

The only symptom that is constant in a brain abscess is headache. Besides this complaint, there is no classical clinical picture that characterizes this disease. The onset may be insidious and slowly progressive as to suggest the development of a brain tumor. A traumatic history, antedating the onset of headaches, may have symptoms of a chronic subdural hematoma. An acute onset and the rapidity with which neurologic symptoms develop may be typical of a cerebral vascular accident. Other acute conditions which an abscess of the brain may simulate vary from that of an acute encephalitis to intoxications of a chemical or systemic character. An illustration of this group was one patient who found relief for his headaches by the mass use of a proprietary headache powder which contained sodium bromide and acetanilide. Not until he developed a papillitis was he suspected of having more than a bromide poisoning, the diagnosis of which had been established from blood bromide studies. In the presence of a febrile illness, rapid in the development of neurologic symptoms and with an increased cell count in the spinal fluid, a diagnosis of acute encephalitis is readily understandable.

The headaches as described by the patients in this series were in many instances of localizing value. The intensity of the distress was to the side of the lesion in the majority of the hemispherically located abscesses. Those lesions within the frontal lobes produced distress, focal to the side in which the abscess was located, but on initial questioning the patients complained of headaches across the forehead area. Many patients with an acute onset described their symptom as being a severe pain topographic to the site of the subsequently demonstrated locus of their abscess. Abscesses located in the cerebellum characteristically had suboccipital headaches and the majority described befrontal distress comparable in intensity to the occipital suffering. Except for the patients with occipital lobe abscesses or those patients with meningitis, none of the patients with cerebral lesions complained of headaches below the nuchal line. Unlike the headaches of brain tumors, the headaches in the majority of these abscess patients was not particularly more severe in the early morning hours of the day. Vomiting was a very common accompaniment of these headaches but was not always projectile in character.

If there is a second characterizing clinical feature of a brain abscess, it is a diminution in sensibility. This may vary from an increase in somnolence, to stupor, to profound coma and finally unconsciousness. The old concept of stupor, slow pulse rate and choked disks as being characteristic of a brain abscess had some clinical foundation but it is to be remembered that any acutely expanding intracranial lesion may result in this pathophysiologic cerebral re-

sponse. The degree of reduced sensibility of any patient with a brain abscess depends on the location of the lesion, the extensiveness of the diseased process, the degree of increased intracerebral pressure that is produced by the disease and the rapidity with which the pathologic condition develops. In general, the more acute the process, the lesser the degree of receptivity.

The subjective and objective symptoms of brain abscesses, aside from the aforementioned ones, depend on the locations of these lesions. A frontally placed abscess usually secondary to a sinus infection, a fracture through one of these sinuses or a penetrating wound in this area may exhibit no localizing handicaps. Others may suffer memory defects, personality changes, motor speech disturbances or indifferences. This last unconcern may be for personal attire, business or family responsibilities or sphincter control. Temporally located abscesses are more subject to generalized convulsive seizures than those located elsewhere in the cerebrum. Visual field defects of a homonomous character are to be found objectively and in right handed persons variable aphasic disturbances are common. Acute abscess formation in the motor or sensory cortex is not likely to exhibit focal convulsions and the illness may be ushered in with an abrupt rapid hemiplegia. This abruptness may be such as to simulate a cerebral vascular accident and fundamentally this may be basically such an accident. The insultant is an infected embolus that becomes located in the middle cerebral artery or its branches. Abscess development within the occipital lobes was uncommon in this series and was secondary to compounded head injuries or to osteomyelitis of the occipital bone. The focal symptoms in one such case were those of visual range defects. It would appear that the cortical responses in brain abscesses are those of a destructive nature and not of an irritative character. Cerebellar abscesses exhibit in general symptoms of reliable localizing value—these being nystagmus, ataxia and incoordination of motor activities; like cerebellar tumors, they most constantly exhibit choking of the optic nerve heads. Of seven patients with cerebellar abscesses, six had such pathologic changes in their optic fundi. One did not; this particular patient had a complete hemiplegia of a flaccid type and no objective evidences of a cerebellar lesion.

The degree of increased intracerebral or intracranial pressure in a brain abscess is as variable as are the clinical pictures. There were twelve patients in this series who had no opthalmoscopic evidence of intracranial pressure increase. One is apt to see the highest degree of swelling of the optic nerve heads with the most extensive retinal hemorrhages and exudative deposits in patients with increased intracranial pressure due to an abscess. In such a patient these fundic changes alone would deter any risks from a spinal puncture. In a few patients the optic nerves were visualized as being normal. The spinal fluid pressure in such a patient may be somewhat elevated but a normal pressure does not exclude an abscess. The variation of the degree of intracerebral pressure is often more impressively demonstrated at the time of craniotomy. This pressure can be the most alarming factor that the neurosurgeon encounters on dural reflection; the only other comparable pressure increase experienced in the operating room is that which one sees in metastatic disease. This high degree of increased pressure is not only an important factor to be kept in mind at the time of the craniotomy but it is more important that one be cognizant of this tension when one undertakes a ventricular estimation or an air injection for localization of the lesion. If an abscess is suspected before instituting these preliminary studies, one should be alert to the possibility of a surgical emergency. When clinical localization is impossible, in spite of an acute risk, accurate information for effective drainage is the choice. A chronic, well encapsulated abscess may have no greater increased intracranial pressure than a similarly sized benign tumor and an air study or ventric-

ular estimation on such an abscess would entail no greater risk than such a study would for tumor localization. In an acute fulminating abscess a simple ventricular drainage may invite a tragic medullary failure.

The most alarming clinical developments can occur in the shortest periods of time in acute abscess formations. The onset may be as abrupt as a cerebral vascular embolic or thrombotic process. This onset occurs with a headache and extensive incapacitating neurologic symptoms appear promptly. If these objective evidences are a little slow in developing, an intracerebral hemorrhage may be suspected. Temperature elevations over and above the elevations of most vascular cerebral insults may be the only clue to a brain abscess possibility. The optic nerve heads may remain unchanged in the beginning of these illnesses and this absence of choked disks serve only to mislead in the diagnosis. Spinal fluid pressures are at a high normal or above. A frank increase in the cells of the spinal fluid is indicative of the infectious activity adjacent to the meninges. This increased cell count with an elevation in the spinal fluid pressure has been the most valuable diagnostic aid in suspecting these acute conditions. Mortality rates in brain abscesses in the future are to be in ratio to the number of these acute "pus containing, diffuse, infective encephalitics" found in any series.

Historic information in years past was likely to be of more diagnostic value than the neurologic examination. An acute otitis media that ceased draining abruptly or one that was complicated by a mastoid infection necessitating a mastoidectomy, was a common lead for a temporal lobe or a cerebellar abscess. A frontal sinus infection, simple or complicated by an osteomyelitis of the frontal bone, was a definite sign of frontal abscess. A history of a penetrating wound of the skull or a story of a skull fracture through the mastoid or the nasal accessory sinuses with a subsequent development of headaches and a febrile course was information sufficient to justify suspicion of an intracerebral infection. Any of these patients today have been administered antibiotics, many of these before any clinical manifestations of infection; the great majority of them have had treatment only sufficient to allay the acute process and allow the more serious sequelae to proceed in their chronic development. Maybe "a little help is better than none" in such a serious potentiality but continued and protracted prophylaxis would be more intelligent therapy. The indiscriminate or minimal use of sulfa and penicillin has clouded more than one of what might have been an early human disaster or a good chronologic medical story of an abscess in the brain. In two autopsies in our series it was concluded that the foci which gave origin to the abscess formation had been so allayed from penicillin therapy that in neither instance was there clinical subjective or objective suspicion and the low-grade chronic process of these infections was only discovered at autopsy. These two experiences are such as to indicate x-ray studies of the mastoid bones as well as the paranasal sinuses in abscess suspects or in operative patients with confirmed abscesses who have had any form of antibiotic treatment. Exclusive of the violently acute abscesses of the brain, a clinical temperature chart today is of no value in the diagnosis of these lesions and can serve as a false safety sign to steer one to other diagnostic possibilities. Should this lead one to a craniotomy in a "tumor suspect," the risks, complications and sequelae with intact or piecemeal total resection of the abscess are not what they once were.

TREATMENT

The initial surgical principle in the treatment of a brain abscess is the clearance of the focus from which the abscess had its development. Whether this is with the protracted use of chemotherapy or antibiotic agents or through radical surgical extripation of this focus or a combination of both is a problem for individual surgical

judgment. If there is a diseased process that gave birth to the brain abscess and this focus is not eradicated, the percentage of abscess cures is materially lessened. Whether the extracerebral pathologic condition is cared for before, during, after or in conjunction with the brain abscess surgery, is again a problem of individual selection. The important fact is to remember that the anti-infectious agents are important adjuncts in the treatment of these infections but are not always to be relied upon to effect a cure, even though the symptomatology might suggest such.

The second surgical principle upon which the successful treatment of a brain abscess depends is accuracy of localization. This is most important, irrespective of the particular type of surgical procedure to be instituted and in spite of the histopathologic phase of the abscess formation. Neurologic, routine radiologic and physical findings may permit precise information as to the site of an abscess. If, however, there is any question of doubt, pneumoventriculography should be instituted. This procedure, as has already been alluded to, may in itself become a very formidable undertaking in certain infectious processes but even in these cases the value of the procedure outweighs its risks, for if the degree of intracranial pathology has attained this serious a level, multiple trephinings and "needling" for the diseased area is a more hazardous procedure than a routine ventricular air study. During the act of localizing the pathologic activity one must decide and plan the surgical attack in keeping with the patient's general condition and duration of his illness; this attack will be influenced mostly by the surgeon's past experiences.

Today, even with a strongly suspected diagnosis of a brain abscess, before any surgical procedure the patient should be well fortified with systemic chemotherapy. If the problem is an acute one, intravenous medication to effect a satisfactory blood level of the sulfa or penicillin, either or both, is the choice of administration. The dosages of these drugs should be of heroic and not homeopathic proportions; in this stage of abscess pathology, the infective organism can be contacted by systemic medication. The question of how protracted this period of treatment shall be is not too difficult to solve in the violently ill patient whose clinical course has been a very rapid one. If such treatment is effective, improvement is likely within twenty-four to forty-eight hours. If these antibiotics are not dramatically beneficial, an increase in the intracranial pressure signs and further reduction in sensibility are the most reliable clinical evidences of a continued, uninfluenced pathologic process. In this acute abscess situation, even surgery is likely to be ineffective but this fact should not influence one against operation; again, "judgment is imperative, time is a factor, and action necessary" if the greater number of these patients are to be salvaged. Decompression alone in our hands has been of no benefit in these patients. Combined with aspiration, a decompression, when the clinical symptoms are more those of increased intracranial pressure rather than sepsis, will be more effective than simple trephine aspiration. It is to be kept in mind that the acute conditions now under discussion are not those in which pus formation is likely to occur. Death in these people is due to the rapid increase in the intracranial pressure and tissue damages incompatible with central nervous system functions. Perhaps in these liquefying cellular destructive phases of the infectious activity, more radical exposures and surgical suction removal of the most evident pathology with the topical use of sulfa and penicillin may prove to be more effective therapy than the conservative efforts of the past. A radical venture no doubt, but certainly the treatment of these fulminating lesions heretofore has been futile. One should utilize one's neurophysiologic knowledge and plan for minor risks of leaving a patient minimally handicapped by such radical efforts.

The treatment of chronic abscess of the brain is easy compared with the treatment

of the acute lesions. When is an abscess chronic? Grossly, when liquefaction and pus formation have occurred, may be a practical answer. The clinical age answer may be one of dispute but certainly a lesion of ten to fourteen days is chronic and beyond this period there are no scientific supports for acuteness other than didactic subdivisions, admitting that within this period acute developments of a secondary vascular character may ensue. As has already been stated these patients are or should be systemically well fortified with sulfa and penicillin therapy. The problem is, "How well is the patient"? If pressure symptoms are subsiding, the septic course suppressed and focal signs are not progressive, these medical therapeutic efforts should not be discontinued in less than eight to ten weeks. In short, as long as the patient is improving no surgical procedures should be instituted. On the other hand, one should not be lulled into a state of false security and rely solely on the antibiotics for a cure of a chronic abscess for there are fewer such cures among chronic abscesses than there are surgical failures. With a total reduction in the occurrence of the disease, it would require a compilation of of many surgeons' experiences to prove this suggestion.

If in a given case of a chronic abscess pressure symptoms demand that one should interfere surgically, a craniotomy should be planned. This should be in accordance with ventricular air depictions of the location of the lesion. A trephine of the skull should be made and a ventricular needle should be introduced in the direction of the already delineated space occupying deformity. If there is a rubbery resistance and it requires some force to introduce this exploratory cannula into this oppugnancy, one has two alternatives. These should be evaluated in terms of duration of disease and the condition of the patient. If the duration of the disease is relatively short and pressure symptoms are dangerous, the osteoplastic efforts of the craniotomy may be performed and in accordance with Vincent and Askenasy's[7] experiences, the abscess may be tapped, the pus evacuated (and penicillin injected) and the surgery discontinued at this place. If pressure symptoms are not too severe, the clinical chart is indicative of no active infectious process and the capsule is profoundly resistant to puncture, the dura may be opened, cortical incision or sacrifice in accordance with the location of the craniotomy exposure and the abscess *in toto*, subsequent to drainage or in piecemeal, may be removed. The technical details of this approach have been previously described.[9]

It is rather likely that King[3] in his cortical marsupialization efforts would not subscribe to his own methods today as a procedure in the treatment of brain abscesses. If the lesion were circumscribed such as to "wander to the surface" after a cortical uncapping, he would likely effect a total extirpation. With effective prophylactic agents against the spread of meningeal infection, it is doubtful that Dowman[2] would be interested today in the production of meningeal adhesions before a surgical attack on an abscess except to allow for chronicity. Coleman, a conservative, having had a string of excellent results from simple drainage, is likely to adhere to the procedures which in his hands have netted the best results. These contributions should be given due evaluation and there are still abscesses, like Macewen's patients, which can be drained through the tract of development with complete results. The choice of treatment for an uncomplicated convalescence and minimal morbidities in the experiences of the author are those abscesses which have been extirpated at the time of craniotomy.

SUMMARY

Antibiotic and chemotherapeutic procedures have tremendously reduced the incidence of brain abscesses and this therapy is indicated irrespective of surgical procedures instituted. This form of therapy in its prophylactic efforts has served in

more than one experience to chronicize and deter diagnostic concepts.

The occurrence of brain abscess formation is still common enough to command the interest of all concerned.

There is no clinical picture that characterizes this disease.

Eradication of the focus of origin of a brain abscess is the first principle in the surgical treatment of these lesions.

Accurate localization of the abscess process is the second most important surgical consideration—a procedure at times not without grave risk.

Total exclusion of the abscess is the surgical goal of treatment and radical efforts to date have been the most effective in this accomplishment.

No cure of a brain abscess should be ascribed in any patient without benefit of either a total surgical extirpation of the lesion or a confirmation of the extermination of the disease by pneumoencephalographic studies.

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DIAGNOSIS OF INTRACRANIAL ANEURYSMS*

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INTRACRANIAL aneurysms are frequent causes of sudden, severe intracranial symptoms. Certainly, the incidence of intracranial aneurysms is much higher than has been suspected in the past. Many patients who succumb from a so-called cerebrovascular accident actually die of a ruptured intracranial aneurysm. Intracerebral hemorrhage with ruptured aneurysms occurs frequently, that is if the aneurysm involves one of the branches of the middle, posterior or anterior cerebral arteries. Aneurysms that actually involve the circle of Willis manifest their rupture by evidence of subarachnoid hemorrhage rather than intracerebral hemorrhage. The incidence of subarachnoid hemorrhage is greater than that of intracerebral hemorrhage because of the anatomic relationship of the larger arteries to the brain. All the larger branches actually lie in the subarachnoid space. Rarely do aneurysms occur in the minute vessels that penetrate the brain substance.

In a personal experience with 114 intracranial aneurysms in 110 patients, the etiology presumably was related to congenital defects in the arterial walls. One exception was a patient with mycotic emboli. Hypertension was noted in twenty-three patients. In none was syphilis a factor.

It is of interest that aneurysms were found in seventy-two females and thirty-eight males. Forty-seven aneurysms were found on the right and sixty-three on the left side.

Unfortunately, there is no clinical syndrome that makes the diagnosis of an unruptured intracranial aneurysm a reasonable certainty. Without evidence of subarachnoid hemorrhage, however, there are certain symptoms which should arouse suspicion of its presence.

That the diagnosis of intracranial aneurysm should be made, if at all possible, before rupture, is dramatically presented by Magee and also more recently by Hamby. Magee found that fifty-two of 150 patients with subarachnoid hemorrhage died in the primary attack. In fifty of the ninety-eighty survivors of the first attack, recurrence took place and thirty-two patients failed to recover. In other words, 35 per cent died in the first attack and 21 per cent succumbed in recurrent attacks. Of the 44 per cent who survived, many were either partially or totally disabled as a result of the cerebrovascular changes. Hamby, in an excellent survey of 130 verified intracranial aneurysms found at the Buffalo General Hospital since its beginning, including autopsy material, reported 39.9 per cent died in the first attack and 28.5 per cent died of recurrent attacks.

Since many of the recurrent hemorrhages take place within three weeks following the initial hemorrhage, it is important that an attempt is made to establish the diagnosis as well as to determine whether or not the aneurysm is located at a site that is amenable to surgical intervention, or whether the patient's chances are as favorable without surgery as with surgery.

The whole problem from the practical standpoint depends entirely on the question as to whether an aneurysm is definitely the cause of the sudden intracranial symptoms. If so, what should or can be done about it? The statistics of Magee, as well as those of Hamby, emphasize the seriousness of the condition. The initial symptom

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of many of these patients was the result of the rupture manifesting itself by a subarachnoid hemorrhage, the patient being normal in every way subjectively before the rupture. There is no possible practical means that one could utilize to uncover an unruptured aneurysm in those patients in whom no previous symptoms were present. Therefore, it is important that patients with symptoms that might indicate an aneurysm should be investigated.

In the surgical experience with 110 patients in whom intracranial aneurysms were verified, the most striking feature was that only nine patients with intracranial aneurysms were encountered in a period of ten years and 101 were found in the past four years, mainly because of an awareness that such a condition might be present. Because of that suspicion, diagnostic aids were utilized either to prove or disprove the presence of an aneurysm. That Dandy had a similar experience is indicated in his monograph on 108 cases of aneurysms which covered hospital reports for a period of fifty years; 40 per cent were necropsy reports and 60 per cent personal surgical experience in twenty years. Thirty-six of these patients were operated on in a period of six and one-half years after his interest had been stimulated in the surgical treatment of aneurysms.

The initial symptoms other than that of subarachnoid hemorrhage in many instances were bouts of unilateral headaches and in others, periodic attacks of generalized headaches. In only four of the 110 patients were typical unilateral attacks of migraine the presenting complaint. Eighteen of the patients had no symptoms previous to the actual rupture. Eight patients had atypical facial neuralgia. Seven patients noted diminished vision as a presenting symptom. In twelve patients the symptoms were initiated by generalized convulsions. Eighty-two of the patients had a history of previous subarachnoid hemorrhage. The symptoms of subarachnoid bleeding may be confused with those of meningitis because of the stiffness of the neck, generalized headache and elevation of temperature. Therefore, one must be alert in taking the patient's previous history to be certain that a diagnosis of previous meningitis might not actually have been subarachnoid bleeding. Forty-five of the patients had a single subarachnoid hemorrhage; the remaining eighty-two patients had two to eight previous hemorrhages. In eighteen patients the symptoms were of gradual onset and were usually caused by local pressure from a large aneurysm in the region of the sella turcica.

Sudden hemiplegia occured in eleven patients. This development in a patient with aneurysm is either due to intracerebral hemorrhage, propagation of thrombosis or an embolus which has broken off the thrombus in an aneurysm.

Periods of dizziness were noted in a sufficient number of patients to justify its mention. In none of these patients was vertigo the only symptom.

Ophthalmoplegia is probably the most common finding in patients with large aneurysms involving the internal carotid artery or in patients with extensive hemorrhage along the third cranial nerve. The latter is especially vulnerable to sudden localized pressure. Since this is true, one cannot be certain as to the exact location or the size of the aneurysm. It is a valuable clue, however, as to the side of the circle of Willis that it involves. Sixth nerve palsy occurred in six of the 110 patients. These palsies also were on the aneurysm side of the circle of Willis, and associated with aneurysms of various sizes with no uniform pattern as to location. Papilledema was found in eight patients; one was associated with subretinal blebs of hemorrhage, pathognomic of subarachnoid hemorrhage. Nineteen patients had optic pallor owing to direct pressure of the aneurysm on the optic nerve.

Involvement of the fifth cranial nerve was present in the large subclinoidal aneurysms, with pressure directly on the first and second divisions. Manifestation of pain in the first and second division of the

fifth cranial nerve was frequently the initial symptom of ruptured aneurysm. The seventh cranial nerve was not involved in any of our patients except for central facial weakness. The functions of the eighth, ninth, tenth, eleventh and twelfth cranial nerves were changed from normal in a patient with a large aneurysm of the basilar artery. In this patient the ninth cranial nerve pressure was indicated by a typical glossopharyngeal tic douloureux.

The usual cause of a bruit in aneurysm is an arteriovenous opening which occurs most frequently by rupture of a subclinoidal aneurysm of the internal carotid artery into the posterolateral surface of the cavernous sinus. The incidence of bruit in aneurysms is low; however, it can occur in patients with localized atherosclerotic changes in the internal carotid artery which have narrowed the lumen of the artery locally. That this can and does occur has been demonstrated in several instances. Partial occlusion of the internal carotid artery in the neck is an example of localized narrowing of an artery producing a bruit or thrill in that region. In seventeen of the 110 patients a distinct bruit was present because of a venous communication with the cavernous sinus. In two patients a direct communication of a branch of the anterior choroidal artery with the vein of Galen caused bruit. Another bruit was elicited in which partial thrombosis had occurred in a large aneurysm of the third portion of the internal carotid artery. In three patients bruit was elicited which could temporarily be occluded by compression of the internal artery. No changes in the branches of the carotid system could be noted by arteriogram, however, on the side on which the bruit was elicited. Two of these patients had aneurysms involving the vertebral artery. In one patient visualization of the vertebral artery and internal carotid artery was normal. Ligation of the internal carotid artery changed the character of the bruit and completely abolished it for a period of a few hours; however, it then returned. The cause of the bruit, therefore, is undetermined.

In eliciting the history of a patient with a suspected intracranial aneurysm, direct inquiry must be made as to whether the noise he complains of is localized. Also, is it synchronous with his heart beat and does its character change with position? If a bruit can be heard with a stethoscope the internal carotid artery may be occluded by digital compression on the side of the bruit. If the bruit disappears completely by occlusion of one internal carotid artery, it is quite certain that it involves the carotid system and not the vertebral arterial system. If the patient complains of a cranial noise synchronous with his pulse which cannot be heard with the naked ear or the stethoscope, a stethotron should be utilized. If this fails to corroborate the presence of a bruit, the internal carotid arteries are alternately compressed. If there is no subjective reduction or cessation of the noise, one can be reasonably certain that the bruit does not involve the carotid system. I know of no method in which a bruit of the vertebral system can be eliminated diagnostically without direct exposure of the artery in the neck unless the artery enters the vertebral foramina higher than usual and possible occlusion is made by digital compression low in the neck.

In any bruit that can be heard equally well over the entire skull which is not due to aneurysm, one must consider advanced Paget's disease or extensive blood vessel anomalies. The presence of a bruit intracranially which occurs suddenly with an association of pulsating exophthalmus means that there is a communication of a large artery with the cavernous sinus unless there is a history of a penetrating injury, such as gunshot and stiletto wounds, or a basilar skull fracture. The fistula between the artery and vein is the result of a rupture of an aneurysm into the cavernous sinus. This occurrence, of course, is lifesaving since it is far safer for an aneurysm to rupture into the cavernous sinus than into the subarachnoid space.

The typical history of a ruptured aneurysm of the anterior two-thirds of the circle of Willis is manifested by sudden onset of a severe unilateral frontal headache, extending rapidly posteriorly, becoming generalized, followed by stupor for a varying period of time—from several hours to two or three days. When the patient regains consciousness, drooping of the eyelid on the affected side is noted with a partial or complete ophthalmoplegia. The generalized headache and stiffness of the neck persists for several days or weeks. Bloody spinal fluid, of course, verifies the diagnosis of a ruptured intracranial aneurysm. It does not, however, indicate the exact site or determine whether or not the aneurysm should be attacked intracranially or indirectly.

From the standpoint of treatment, the importance of verification of the diagnosis cannot be overemphasized so that one is in the position to advise the patient and family intelligently. As to the treatment, the surgeon must know the exact site of the aneurysm; he must know the tolerance of each cerebral hemisphere to reduction in blood supply (collateral circulation); he must know the risk involved in nonsurgical treatment versus indirect or direct surgical intervention; he must know statistics as to the time a secondary hemorrhage is likely to occur; he must know whether the aneurysm is located where its removal is likely to cause permanent hemiplegia. For this reason, arteriography[5] plays a most important part in the problem of aneurysms. Arteriograms in most instances are imperative both from the standpoint of the presence of an aneurysm as well as to determine its exact location, allowing one to judge from previous experiences as to whether its location is relatively safe for direct intracranial attack by excision or trapping or whether the indirect method by proximal ligation in the neck would be the safer of the two procedures.

The plain stereoscopic x-rays may give valuable information as to the presence of

Fig. 1. Sizable subclinoidal aneurysms have a tendency to erode the anterior clinoid upward or sharpen it on the inferior border. Since the majority of aneurysms are in the region of the circle of Willis, the roentgenograms should be closely scrutinized in the region of the sella turcica for calcification.

aneurysm. (Fig. 1.) Only the large aneurysm which is in direct contact with bone can possibly cause changes with the exception of possible calcification which may occur in an aneurysm. It is usually manifested by a fine crescentic outline or dense calcification (Sosman and Vogt). Thinning of the lateral margin of the optic foramen has been described by Jefferson.

The final diagnosis, therefore rests on, either the arteriogram or direct surgical exploration. In my experience, arteriography has been of great help, allowing one to study the relationship of the aneurysm to the main branches of the circle of Willis. For that reason, arteriograms are now made in all patients suspected of having an aneurysm. Over 200 arteriograms have been made in most cases with thorotrast; however, more recently diodrast has been employed with no untoward results.

The question arises, when should arteriograms be instituted in patients with recent subarachnoid hemorrhage? I have no

Fig. 2. Equipment necessary for injection of carotid artery by the indirect method.

hesitation in injecting thorotrast or diodrast at any time if a No. 18 gauge needle is used since any increased intra-arterial pressure caused by the injection would be dissipated before reaching the site of the aneurysm. Many times it is impossible to determine from the physical examination or history the side on which the aneurysm is situated; therefore, both carotid arteries and their branches should be visualized by the closed method so that the proper site of the leaking aneurysm may be determined before surgical interference is instituted.

The ligation of an internal carotid artery can be of no value in an aneurysm which does not involve the anterior two-thirds of the circle of Willis. Therefore, in a non-localized bleeding aneurysm, if no aneurysm is disclosed by bilateral carotid arteriography, the vertebral arteries with their branches should be visualized.

The choice of the contrast media should be made on the basis of whether the patient is sensitive to iodine. At the present time I use 35 per cent diodrast in all patients who show no evidence of reaction to a test dose of diodrast. Thorotrast is used whenever there is any question of sensitivity to iodine. Diodrast is preferable, even though the contrast is not as sharp as with thorotrast since the injection may be repeated several times if for some reason the arteriograms are unsatisfactory, whereas 20 cc. of the thorotrast is about the maximum volume one should use in any given patient, and preferably 10 cc., because it is not excreted.

The method of injection is, of course, important. At present I use only the closed method since it may be employed in patients with equivocal signs of aneurysm in whom the air studies are normal. Arteriograms can be made with the patient still asleep from pentothal anesthesia, immediately following the review of the encephalogram. In patients with evident manifestations of aneurysm one is perfectly justified in making a direct exposure for the arteriograms. The reason I do not use the open method routinely is that one is inclined to ligate the internal carotid artery in the neck as soon as the aneurysm can be seen on the wet x-ray film. Adequate study may not be made of the exact site as to its relationship and involvement of other branches of the circle of Willis. If a direct attack seems indicated, one should allow as much blood supply to remain as possible, and therefore ligation in the neck is left undone. In other words a more deliberate plan of treatment may be followed. With normal air studies, vascular changes have not been excluded. For that reason the indirect or the so-called closed method of arteriography is an added minor procedure, whereas exposure of both carotid arteries verges on the major, nor is one assured of better filling with the open than with the closed method. Incidentally, many more aneurysms will be uncovered by performing an increased number of arteriograms. I am, therefore, convinced that the combination of encephalograms and arteriograms should be used more frequently than at present; that is, if the encephalogram shows no gross changes, this procedure may immediately be followed by the closed

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Fig. 3. Relative position of patient and operators to the x-ray equipment.
Fig. 4. Position of patient on table to prevent overextension of neck during injection.

method of arteriography under the same anesthetic agent.

The technic of arteriography is simple, even though it took me a long time to develop a method that produced satisfactory arteriograms. It is important to keep in mind that the completion of a satisfactory arteriogram depends on the teamwork of the operator, the x-ray technician and, of course, the physical equipment, namely, the x-ray apparatus and the syringe and needle which must be in perfect working order. (Fig. 2.)

The patient is allowed to remain on the carrier stretcher on which he has been transported to the x-ray department. (Fig. 3.) The wheels of the carriage are firmly fixed so that no motion can take place at the time of the injection. With the patient on his back, a pillow or folded blanket is placed underneath his shoulder blades and two folded Turkish bath towels are placed under the occiput. Care must be exercised to prevent overextension of the patient's neck, either by lifting up the chin during the pentothal

anesthesia or by allowing the head to rest considerably lower than the chest. (Fig. 4.) It would seem probable that, by extension, the large vessels of the neck would be held on a stretch, thus allowing the needle to enter the lumen more readily.

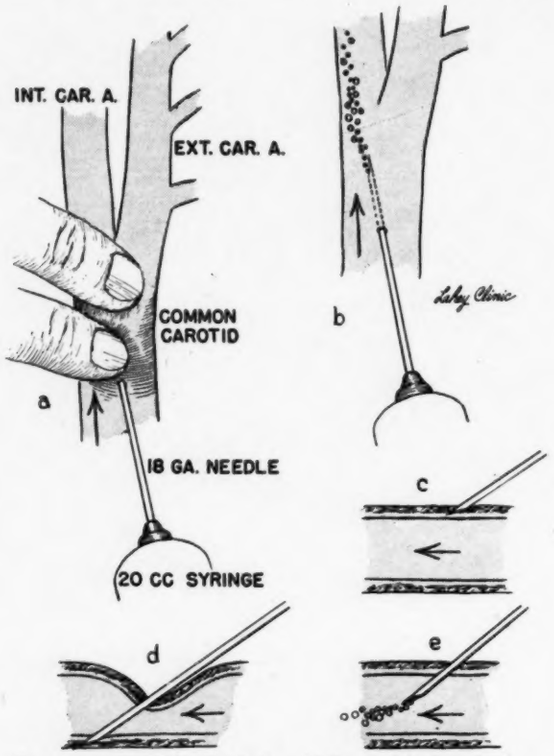

FIG. 5. *a*, Position of left middle and index fingers; the middle finger is over the carotid bulb; the index finger is over the common carotid artery. The point of the needle engages the common carotid immediately beneath the index finger. *b*, Demonstrates "streamline flow" of the contrast medium. *c*, Sharp-pointed, curved, beveled needle engaging adventitia. *d*, Needle withdrawn slowly so that it is well in the lumen of the artery. *e*, Compression of arterial wall by the needle, indicating the reason arterial blood may not enter needle.

The opposite, however, has been my experience. The lumen of the artery is actually narrowed and may in some instances be occluded by the stretch over the tubercle of the sixth cervical vertebra. The patient's head is allowed to remain in a horizontal plane. The index and middle finger of the left hand are used to palpate the common carotid artery, the middle finger (Fig. 5) is allowed to rest on the carotid bulb and the index finger on the common carotid artery beneath. A No. 18 gauge needle, 2 inches long, with a very sharp point and moderately curved bevel, attached to a 20 cc. Luer-Lok syringe by a two-way petcock, is directed so that the point of the needle engages the adventitia of the common carotid artery immediately beneath the site of the index finger. Delicate pressure is then exerted on the needle with the syringe, and immediately released if there is any tendency for the artery to roll to one side, since this means that the point of the needle is engaged eccentrically and not dead center. When one is certain that the needle is dead center over the artery, which must be noted entirely by the feel of the index finger of the left hand and the transmitted feel of the needle through the syringe to the right hand, the needle may then be inserted into the artery, arterial blood can be seen pulsating into the citrate solution which is in the syringe, and it has a tendency to push the piston of the syringe outward. If arterial blood does not enter the syringe, the needle is slowly withdrawn, since many times the arterial wall has become constricted at the site of entrance by the pressure of the needle and, as one withdraws the needle slowly, the artery expands and arterial blood may be seen pulsating into the syringe. By careful manipulation one can then place it well into the artery. The next step is to be certain that the needle is well engaged in the lumen of the artery and will not be displaced by the injection. It is well to take several practice injections with the citrate solution which is used to prevent clotting of blood in the syringe and needle. If several cubic centimeters can be injected rapidly and a free back flow of arterial blood takes place, with the needle directed toward the lateral wall of the common carotid artery, the syringe with the diodrast is then attached and the solution injected as rapidly as permitted through the No. 18 gauge needle. Great care must be exercised to prevent bubbles of air, which become caught in the hub of the needle as the

syringe is attached, from being injected into the carotid system. To prevent this air from being injected, blood can be allowed to flow into the contrast medium so that the two or three small bubbles of air will be carried to the upper portion of the syringe and remain there during the injection. Certain serious sequelae can take place from air emboli and may account for reported convulsions occurring during injection.

It is important to inject the contrast medium toward the lateral lumen of the common carotid artery because a definite law of "streamline flow" holds true in the arterial system. In other words, if the contrast medium is injected laterally and downward, it will follow the blood stream into the internal carotid artery; whereas if the contrast medium is injected medially and upward, most of it will appear in the external carotid artery.

It is necessary that the x-ray technician be made aware that the injection is about to start. The x-ray tube is prepared for making the exposure. The head is thoroughly aligned with the upright automatic Bucky, suitable for stereoscopic exposures, which is used routinely for chest roentgenograms, 20 cc. of the dye has been placed in a 20 cc. syringe and 15 cc. is injected rapidly, at which time the first exposure is made. Then the injection is continued during the automatic shifting of the x-ray tube and the films for the second exposure. This allows adequate filling for stereoscopic films and in most instances the latter are more satisfactory than a single lateral view and an anteroposterior view. (Fig. 6.)

A word of caution should be inserted here that the operator and assistant must be careful not to allow themselves to be exposed to x-rays beyond the dosage that is safe for their own welfare. The dosage that can be tolerated may readily be computed by the roentgenologist and one should abide by his judgment.

Aneurysms of the posterior third of the circle of Willis fortunately occur in only

Fig. 6. Arteriogram with aneurysm involving a vestigial artery, the rupture of which caused complete third nerve paralysis. The anteroposterior arrow points to the internal carotid artery, the posteroanterior arrow to the aneurysm. The arteriograms taken stereoscopically afford an excellent method of studying the relationship of the aneurysm to the main arterial branches.

25 per cent of all aneurysms involving the intracranial cavity and can be visualized by arteriography of the vertebral arterial system. Unfortunately, the closed method of injecting the contrast medium is more difficult; however, it can be executed in individuals with a high subclavian arterial arch. Direct exposure of the vertebral artery can be done. The law of streamline flow in the vertebral system is of importance in that the opaque medium follows the blood flow from the vertebral artery into a common reservoir, the basilar artery, filling only the branches on the side injected as well as the posterior cerebral artery on that side.

COMMENT

The diagnosis of intracranial aneurysms should be made before intracranial catastrophe takes place if at all possible.

An awareness of the frequent occurrence of intracranial aneurysms and of the fact that many of these patients may be helped surgically should stimulate us to institute diagnostic means that may reveal the aneurysm before actual rupture occurs.

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Following trauma to the head intracranial bleeding of four different types may occur. Three types involve the brain only indirectly by pressure or irritation. When an extradural hemorrhage is suspected, an exploratory burr hole should be made in the mid-portion of the temporal bone just below the insertion of the temporal muscle. The skin incision should be outlined so that the initial incision can be converted into a subtemporal decompression.

INTRACTABLE PAIN DUE TO CANCER

TREATMENT BY NEUROSURGICAL METHODS

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Denver, Colorado

NATURE OF PAIN DUE TO CANCER

STRANGELY enough cancer is not inherently a painful lesion. Pain is due to secondary or neighborhood effects. Of chief consideration are obstruction of a viscus, infiltration of nerves, compression of nerve roots or trunks against bony structures, infiltration, tumefaction and swelling in tissues snugly invested by fascias and rich in nerve trunks and plexuses and lastly infection and inflammation. Obstruction at some locus in the gastrointestinal tract is illustrative of the first classification. Carcinoma of the stomach, sigmoid colon and rectum are relatively painless until the symptoms of obstruction supervene. Inasmuch as obstruction is amenable to general surgical procedures, the neurosurgeon will seldom see those patients before metastases or extensive infiltration has occurred. Infiltration of the brachial plexus and nerves in the axilla is common in patients with metastasis from breast cancer. Compression of nerve roots is manifest in metastases to the spine. Metastases to bone are relatively painless unless there is some gross distortion or involvement of nerve roots. Witness the painlessness of metastases to the skull. Metastases to the spine from carcinoma of the uterus, breast and rectum are often painful and one finds bulging and stripping of the dura with crowding of nerve roots at their intervertebral foramina. Seldom do such metastases transgress the dura early to become intradural, a point worthy of mention. Metastases which are late in manifestation and which grow slowly may at first cause pain in a single root distribution.

The presence of a primary carcinoma may not be suspected or the significance of one treated some years before may be over looked. The following case is illustrative: N. R., a white woman of seventy-one, was admitted to Mercy Hospital in Denver on January 16, 1945. She complained of severe pain in the distribution of the left occipital nerve. Her history revealed that a carcinoma of the breast had been removed five years ago but since careful examination, including roentgenograms of the skull and cervical spine, were negative a diagnosis of occipital neuralgia (inflammatory?) was made. The persistent and severe nature of the pain prompted avulsion of the occipital nerve on the left. This provided some relief for a short time. She then went through a period of vestibular disturbance that was taken to be labyrinthitis and it was not until two years after avulsion of the occipital nerve that involvement of the ninth to the twelfth cranial nerves on the left, with hoarseness and dysphagia, betrayed the true nature of the pathologic condition. A roentgenogram of the skull now revealed punched out areas near the foramen magnum. In a somewhat desperate attempt, at the patient's behest, the nerves were explored. The ninth, half the vagus and the second and third cervical sensory roots were cut. Metastases had grown *en plaque* about these nerves extradural but had not become intradural. During the month that she lived after operation, metastases grew rapidly. Tumefactions raised over the skull with incredible rapidity.

The significant lesson is that the agonizing pain which she suffered for almost two years might have been treated intelligently had the probability of obscure metastases been given due consideration.

The pain notoriously associated with carcinoma of the uterus and uterine cervix is undoubtedly due to infiltration of tissue planes in the pelvis with consequent infiltration of the lumbosacral plexuses and compression against the bony pelvis.

The anatomy of the neck is characterized by closely invested tissues rich in nerve trunks. Tumefaction and induration involving tissues of the neck are characterized by pain and tenderness.

When cancer erodes mucous membrane or skin, infection and inflammation develop and progress deeply into the lymphatics and fascias. Carcinoma in and about the buccal cavity is the outstanding example especially when infiltration involves the throat and neck. As an exception, carcinoma of the lip even with metastases to regional nodes is not always painful.

Hence, from this brief resumé it might be conjectured that neurosurgery is enjoined largely for advanced cases of carcinoma of the uterus and cervix, breast and buccal cavity. Ninety per cent of the author's chordotomies have been performed for pain of carcinoma of the uterine cervix with pelvic infiltration. In the minority group are a few patients with carcinoma of the rectum with metastases to the spine. The remaining chordotomies were performed because of lesions other than cancer.

TREATMENT OTHER THAN NEUROSURGICAL

While it is not entirely within the province of this paper to discuss other methods of treatment, those most commonly employed are mentioned briefly for orientation and because they have such an inextricable bearing on the subject. Toward this end, narcotics, radiation and sex hormones deserve some discussion.

Narcotics. Morphine is naturally the first court of appeal when pain becomes a real and persistent symptom. When pain becomes a symptom of inoperable cancer, it is usually persistent and the attending physician has little else at his disposal but this greatest of all pharmacologic blessings to meet the exigency and ease the pain of his patient. The administration is continued for varying periods until the inevitable threat of addiction becomes manifest or until the drug becomes inefficacious. The young and enthusiastic neurologic surgeon is likely to underestimate the demanding and natural circumstances under which the drug is administered by the family physician and to place too great an emphasis on the evils of addiction and its contaminating influence on the success of surgery. Not all patients with the cancer-pain syndrome are appropriate candidates for surgery. If a patient, after radiation, can be made relatively comfortable with the drug throughout his limited days, the author knows of no superior treatment. There are exceptions, examples of which are given in this paper, and it is not easy to exercise perfection in judgement in the given case. The "magic drug"* would be the treatment par excellence for it stands to reason that surgery designed to relieve intractable pain only, for one whose time is short, is a major, last resort undertaking and an indication of the dire need for a less trying and less expensive management.

Radiation. It would be presumptive in so short a space to attempt to give due credit to this fascinating branch of medicine in which the energy of electromagnetic waves is enjoined to destroy abnormal flesh and preserve the normal. Of the limited contact and experience of the author, the following convictions have conditioned his judgement and practice:

1. The effects of radiation upon cancer pain are unpredictable but beneficial effects prevail by such a wide margin that an honest effort to use it must be encouraged. When properly administered, harm is so minimal that it can be discounted. The prospective benefits are two-fold: possible relief of pain and attenuation of growth propen-

* At the time of writing a new drug," dolophine," is being carefully dispensed under controlled management. It has the analgesic properties of morphine with minimal powers of addiction. This field of research is most intriguing and urgent.

sities of the neoplasm. Relief of pain is often quite satisfactory for a time although unfortunately it is not often permanent. It is, nevertheless, a gain in both time and courage and the administration is in no way an ordeal for the already tired, weary and anxious patient. The immediate relief of pain and quieting effect of radiation on bone sarcoma, for example, has been very impressive to the author.*

If radiation does not cure, it certainly attenuates the new growth to varying degrees and reduces toxicity with systemic improvement. Occasionally, a very happy and remarkable cure ensues. Cooper and Archer,[1] to cite an example, report the case of a six year old child appearing *in extremis* from metastasizing Ewing's tumor of a rib. Half-hearted radiation resulted in a cure. They emphasize that radiation often relieves pain due to bone metastases.

2. The author does not believe that this branch of therapy has reached its limitations and when considering the desperate nature of inoperable cancer what other effort can be better recommended? Perhaps the fact that wave physics and clinical medicine are in practice, such full time and divergent fields has delayed progress that may yet be attained. He also believes that the domain of roentgenology, particularly that of radiation, has been well manned by conscientious and progressive disciples. Gratifying reports are not often overdrawn.

In short, as a matter of policy and with very few exceptions, the patient with inoperable carcinoma and pain should be turned over to the radiologist at least for the benefit of consultation and certainly before undertaking neurologic surgery.

Sex Hormone Therapy. While this subject is in its infancy, it is of sufficient importance to be listed here. Adair[2] reports that after about two weeks of testerone therapy the relief of pain from bone metastases of breast cancer is usually very real. Huggins, quoted by Adair,[2] reported cures as high as 25 per cent in patients with cancer of the prostate. In the discussion of a paper by Crutchfield,[3] Archer states that patients having carcinoma of the prostate associated with pain should have the benefit of orchiectomy. The relief of pain is dramatic in many instances.

NEUROSURGICAL APPROACH

The selection and preparation of patients for neurosurgery should be made with considerable perspective and discrimination. This is said because if neurosurgery is to be effective, it must often assume the proportions of major surgery. Neurological deficits are likely to result as side effects when incisions are made into the mesencephalon, medulla or spinal cord.

As already stated, and except for some special reason, the patient should be a victim of *intractable* pain. Radiation should have been given a fair trial. Neurosurgery should be considered the last court of appeal but it is by no means implied that it should be delayed unnecessarily.

Due consideration should be given to three aspects of the undertaking: the neoplasm, the pain and the patient. In respect to the neoplasm it is almost embarrassing to insist that the diagnosis of inoperable malignancy should be unassailable. Fortunately, mistaken diagnoses are not common because histologic confirmation has become almost universal practice and by the time a cancer is considered inoperable it usually is. Diffuse pain in the pelvis, low back, in the bladder region and with some radiation in the distribution of the lumbosacral plexus on one or both sides,

* Relief of pain is probably accomplished because radiation quenches inflammation thereby shrinking the mass and relieving tension. This may be the only pain relieving property of x-rays. If so, one might make prophesies in relation to a given lesion and explain the limited duration of relief from pain by radiation. It is probably true that constant and certainly pulsating pain from any tense swelling is due to a stretch of nerve fibers. If tension is diminished, as in the letting of pus, pain abates.

are hardly to be mistaken in respect to their ominous significance if they develop some time after a proven carcinoma of the uterus. The type of neoplasm, its grade of differentiation and other characteristics which help to predict its life history are pertinent. On the whole, a highly undifferentiated cancer that is rapidly growing and taking its toll of the patient in the form of cachexia and mental torpor might be more wisely treated by narcotics. There is no virtue in expediting neurosurgery two weeks before the patient dies. On the contrary, and as a general statement, a scirrhous type of cancer may, with aid of radiation, remain relatively quiescent for several years and if the patient is free of pain show little effect upon the general system. Thus, a young woman (G. B.) was admitted to the University Hospital in Iowa City in December, 1941, with severe intractable pain in the pelvis and lower extremities resulting from inoperable carcinoma of the cervix. Bilateral section of the spinothalamic tracts at the first dorsal segment abolished pain completely. Although she appeared frail and exhausted, we realized that this had been due more to pain than carcinoma. With the will to live, she was discharged in three weeks having gained weight and presenting relatively little untoward neurologic deficits. She joined her husband who was in the service and lived for five years without pain and to all appearances in good health. The break in health was then rapid. The tumor throughout its course was hard and nodular to palpation.

Experience teaches that pain may be overestimated by some patients. Some may be inclined to over-rate the benefits of surgery and not until they are thoroughly apprised of all factors do they conclude that the pain which they have is satisfactorily amenable to simple drug therapy. While it often requires only minutes to be convinced of the realistic and severe nature of pain and the justification for early surgery, there are equivocal situations in which it is well to visit the patient a number of times before coming to a decision. By thus becoming better acquainted with the patient and observing his reactions, the author has forstalled operations that proved better left undone.

Any discussion concerning the patient should be prefaced with the fact that the purpose of the type of surgery under discussion is strictly humanitarian. The objective is not only to eliminate the sensation of pain but to help the patient feel as generally contented as possible. Toward this end the surgeon should become acquainted with him, instilling confidence and establishing rapport. While firmly discussing the possible sequelae of operation, the unpleasant features can be minimized and relative values made clear. These practices have on occasions prepared the patient and allayed anxiety so that he was contented and at peace although the lower extremities were weakened and the bladder atonic. In short, it is poor policy to attempt in any way "to sell" the operation to those patients. When the facts are placed before them with clarity and human rapport, their election should be respected as the best judgement.

OPERATIVE METHODS

In electing some operative procedure to relieve intractable pain, the author has become convinced that the "all or none" policy is best. When the decision is made after proper deliberation, a procedure designed to give optimum and certain results should be carried out. Lesser and "more conservative" procedures or "trials" and multiple operations only torture a weary patient and nullify confidence. The author has concluded, therefore, that except for pain in the face and throat, a section of the spinothalamic tract at an appropriate locus is the procedure of choice at the present writing.

Intraspinal subarachnoid injection of alcohol, various types of sympathectomy and rhizotomy have a very limited application.

Subarachnoid Injection of Alcohol. In my experience the subarachnoid injection

of absolute alcohol has been of little assistance in treating the pain of cancer. This is because the great majority of patients seeking aid have diffuse pain in the pelvis and lower extremities, involvement of the brachial plexus or pain in the head and face. The first two categories are best treated by other methods than deafferentation and the third category does not lend itself to subarachnoid injection of alcohol for obvious reasons. The use of alcohol is practically limited to the distribution of the thoracic nerves and finds wider application in the treatment of pain from other causes than cancer. Disadvantages in its use may be listed as follows: It is difficult to obtain the wide zone of analgesia that is usually necessary. With 1 to 2 cc. of absolute alcohol one may expect to eliminate the nearest sensory root and partially eliminate the two adjacent roots. It is precarious to inject alcohol in the region of the conus medullaris. The origin of the lumbosacral nerves is concentrated here and some paralysis of the lower extremity associated with paralysis of the bladder sphincters almost invariably results. To illustrate the limited but valuable application of a method of this kind, I recall a patient with carcinoma of the bladder and severe pain referred to the end of the penis. The patient was placed in the prone position with buttocks elevated and 1 cc. of absolute alcohol injected into the subarachnoid space at the L-5 interspace. The relief of pain was immediate and permanent and since the bladder sphincters were already paralyzed this constituted no hazard.

Rhizotomy. This is the treatment of choice for pain in the face, tongue, mouth and throat. Any further application of deafferentation, as Bronson Ray[4] points out, is limited to pain in the neck and trunk. He does, however, recommend rhizotomy in respect to roots of the brachial plexus and partial rhizotomy in selected patients. Excluding the head, cancer pain almost always involves the brachial or lumbosacral plexuses to some extent. Hence, neurosurgical treatment must take these territories into consideration. Aside from the unsatisfactory zone of the shoulder and upper extremity I have in recent years entertained no equivocation in regard to the choice between rhizotomy and chordotomy for cancer pain.

Sympathectomy. Insofar as I know, sympathectomy alone has no real application in treating cancer pain. Very little is known about sympathetic pain bearing afferents except in relation to the viscera. Even here I use the term *sympathetic afferent* with reservation since many anatomists still avoid the connotation and prefer the term *visceral afferent*. The weight of opinion eases them into the category of somatic afferents functionally although they do course through the sympathetic chain. In any case, whatever the sympathetic factor may be, it probably never exists alone in our cancer patient and it is expunged along with the somatic factor by either an appropriate rhizotomy or section of the spinothalamic tract. Deference should be given to one possible exception, that of presacral neurectomy. This has a limited application in the treatment of certain kinds of pain emanating from the uterus and distal portion of the bladder. I have yet to see a case of cancer pain in which it could with any reason be proposed as a single procedure.

I have noted a tendency for some surgeons to regard presacral neurectomy as a cure-all for pain in the pelvis. Even though the neurectomy were actually accomplished through the hazards of a carcinoma-infested pelvis, it constitutes only a futile operation.

OPERATIVE PROCEDURES OF CHOICE

By and large and from an operative standpoint, the neurologic surgeon is called upon to relieve pain in one of three corporeal territories: (1) The face, mouth, tongue, throat and neck; (2) the shoulder and upper extremity and (3) that part of the body from the level of about D-5 down. In the first case, surgical elimination of the transmission of pain has been developed

about as far as anatomy permits. Neurologic surgeons are at relatively little variance concerning the general method of treatment. If pain is limited to some distribution of the trigeminal nerve, one may transect the root of that nerve by the subtemporal or occipital route. Sjöqvist,[5] in 1938, proposed a method of dividing the descending tract of the fifth nerve in the medulla. It will be recalled that as the sensory fibers of the fifth nerve enter the pons, a part of them course upward as the upper root and a large proportion course downward in the descending or bulbospinal tract. The tract extends as far downward as the second cervical segment. The fibers in this tract are axons of primary sensory neurones and synapse with secondary neurones in "the nucleus of the descending tract." The descending tract, therefore, is comparable to Lissauer's tract in the spinal cord and the nucleus of the tract is comparable to the substantia gelatinosa in the spinal cord. The fibers in the descending tract appear to be concerned with pain and temperature only. By inference the fibers in the upper root evidently transmit touch. Hence, while a section of the descending tract constitutes a section of primary neurones just as does section of the fifth root, the former has the decided advantage of eliminating pain with preservation of touch. Sjöqvist proposed that the section be made in the medulla at the level of the lowermost rootlet of the vagus nerve. The chief disadvantages of the procedure are injury to the restiform body and the fact that such sections always entail a certain amount of guesswork. The latter may be controlled by skill and practice and should not defeat a method which has merit. The former has been obviated in sufficient measure by making the section 10 to 12 mm. lower than the locus proposed by Sjöqvist. This modification in Sjöqvist's operation has been proposed and practiced by Grant and Weinberger[6] (1941) with no significant difference in the extent of resulting analgesia. In 1943, Grant[7] reported the results of thirteen medullary tractotomies by this method for the relief of cancer pain in the face. He recommended the method as a more desirable alternative than fifth root section for cancer pain not only in relation to its selectivity for pain and preservation of corneal integrity* but because in his experience there has been almost uniform absence of postneurectomy paraesthesia. (In the surgical treatment of tic douloureux, Grant's results have not been such as to warrant adoption of the modified Sjöqvist operation.)

The failure of the Sjöqvist operation to relieve the painful paroxysms of tic douloureux at first appears inconsistent in relation to the facial analgesia obtained. The failure, however, has been common experience. I believe that it may be explained by the fact that the intraoral zone is not made totally analgesic. I have given evidence[8–10] that demonstrates a strict relationship between the trigger zones (predominantly oral and immediately perioral) and the painful seizures. When the oral zone is made analgesic, the tic pain is abolished regardless of the referred distribution of the latter and if the trigger zone is not made analgesic the tic pain will not be cured. It may be recognized that the following statements have a general conformity with this proposition:

(1) As one severs the fibers of the fifth root from below in an upward direction (mandibular toward ophthalmic topography), anesthesia begins intraoral and progresses in a manner suggesting a segmental distribution but combined with some characteristics of peripheral branch distribution.[8] Hence, a partial section including the so-called maxillary-mandibular fibers by rendering the buccal zone analgesic (anesthetic?) abolishes the tic pain.[9,10] This is, no doubt, why Dandy "opined," as Sjöqvist put it[5] that the pain fibers in the fifth root near the pons were congregated near the operator. Obviously, such could not be the case or pain sensibility would be differentially lost in the entire distribution of the fifth nerve after partial section. It is the oral or "trigger zone" fibers that are nearest the operator. In personal

* In Sjöqvist's patients, corneal sensibility was greatly diminished or lost. This would seem logical because the medullary section eliminates pain sensibility most thoroughly on the ophthalmic side of fifth distribution and corneal sensibility is understood to be exclusively pain.

conversation Dr. Dandy said he meant these fibers in question to be "the pain fibers of tic douloureux." Dr. Dandy had characteristically made an accurate and significant observation leading to a correlation between partial section and tic pain, but I do not believe he had yet recognized the probable implication of the trigger bearing zone. Sjöqvist concludes from studies on degeneration of the tract after dividing root fibers that, "The portion of the root which is cut at the "Dandy operation" corresponds to the dorsal (or superior) part of the tract and nucleus. This portion consequently must be formed by the maximillomandibular fibers."

(2) As one destroys the descending spinal tract of the fifth nerve in a caudal to a cephalic direction, analgesia progresses in a strictly segmental fashion toward the buccal zone and the intrabuccal zone is the last segment to be affected.[11,10] It is, therefore, represented by the highest portion of the descending tract of the fifth nerve. This is unfortunate because medullary section of the tract cannot feasibly reach high enough to definitely include the intrabuccal zone. In short, medullary section severs all fibers of the fifth nerve except the appropriate ones in relation to "tic."

(3) The results that Sjöqvist[5] demonstrates in his patients bear out these facts. He did not often mention the effects on intrabuccal sensation. In the one patient (Case IV) in which he did mention it, however, there was "no impairment of the tongue." This was a case of most complete analgesia elsewhere, the section being at maximal height (midolivary). Moreover, corneal sensation was always markedly diminished or lost. This is not only an unfortunate result of the operation but is in keeping with the fact that the section eliminates thoroughly the ophthalmic zone.

Partial section of the fifth root by the subtemporal approach is, at present, the optimum operation for tic douloureux. In approaching the root extradurally, (the Frazier operation) a transient facial palsy has occurred in one of three to five patients. In two patients it has been total and permanent. It has made its appearance as long as five days after operation. It is somewhat of a mystery but it is attributed to injury of the seventh nerve through avulsion of the greater superficial petrosal nerve when the dura is stripped. By approaching the root intradurally and exposing it through a window in the overlying dura the "bug-bear" of facial palsy has been totally obviated. The operation is actually less difficult and the approach almost bloodless. The results now leave very little to be desired.

In the case of pain in the face and throat, the ninth cranial root should be cut in addition to that of the fifth, or the alternative tract section just described. The fact that a ninth root section and a modified Sjöqvist operation can be done through a relatively small exposure in the posterior fossa and without great technical difficulty constitutes a distinct advantage. Moreover, by extending the central limb of a unilateral occipital incision (or the central limb of a cross bow incision) the posterior roots of the second, third and fourth cervical nerves may be severed for pain in their distribution.

Pain in the shoulder and upper extremity is in the large majority of cases an outcome of inoperable malignancy of the breast. Metastases to regional nodes, displacing and encasing the cords of the brachial plexus with eventual swelling, induration and lympho-edema of the shoulder and upper extremity associated with tenderness and severe pain is a pitiable situation. The plea for relief is an inescapable challenge to the neurologic surgeon. This territory is still unfortunately a sort of "no man's land." Neurosurgery has not yet been sufficiently adapted to the treatment of this zone and the surgeon is less confident than he is in respect to the other two corporeal territories. Section of the brachial plexus or intraspinal section of the sensory roots of the brachial plexus, even if such should eliminate the pain, presents the spectre of a dead and paralyzed extremity. In my experience, most of the patients whose mental and physical states were otherwise intact had great difficulty in resigning themselves to this ever present objective evidence of mutilation and the result fell short of being commendable. To make an issue of cutting only the sensory roots and preserving the motor is little short of quibbling. The fact that man can move a deafferented extrem-

ity on request, has in my experience provided little compensation and no consolation to the patient. There is something horrifying and gruesome about a totally dead upper extremity that dangles as an ever present senseless appendage. This reaction is far more intense in respect to the upper than to the lower extremity. It is granted that when there is no alternative, such a result is less an evil than a painful extremity which is just as useless. However, the rationalizing of those who do not have to swallow the medicine makes it taste no sweeter for those who do. Since our premise is to eliminate pain with minimum neurologic mutilation and melancholic despair, we are moved to search for a better alternative than deafferentation.

I have implied that even after section of brachial plexus roots or cords, pain in the extremity may not be abolished subjectively. I have encountered this paradox a number of times not only in respect to a deafferented upper extremity but in respect to other anesthetic zones. For example, M. S., a white woman of middle age, was admitted to the University Hospital, Iowa City, in December, 1941. She complained of severe pain in the right upper extremity as a result of inoperable carcinoma of the breast. The posterior roots from C-3 to D-1 inclusive on the right were cut. The extremity was totally anesthetic to objective tests but the patient continued to refer pain to it. Stroking the upper right chest with ones finger tips or certain places on the scalp or even stroking the sole of the right foot caused "tingling" sensations referred to the hand of the deafferented extremity. Because of pain in the axilla and upper right chest and the phantom pain in the extremity, anterior chordotomy was performed at C-8 on the left. After this the pain in the extremity largely disappeared but the phantom tingling persisted.

The first solution to this problem of the shoulder and upper extremity that would naturally suggest itself is section of the spinothalamic tract at a high cervical level. My experience with this procedure has been disappointing on two counts. First, the level of analgesia has not reached high enough to satisfactorily relieve pain in the shoulder and upper extremity and second, the postoperative course has been stormy and beclouded with confusion, anxiety and respiratory embarrassment.

In relation to the level of analgesia, the author came to the following reasoning. After an experience with many anterior chordotomies at the level of D-1 (the sections being made between the eighth cervical and first dorsal roots) he concluded that the level of effective analgesia could never be brought higher than the skin level of D-5 by this method. Often the level of effective and total analgesia reached only to D-6. Hence, there was always an overlap zone of four to six segments between the level of cord section and the skin level of effective analgesia. Assuming that this same discrepancy in levels would apply to the cervical cord, it is clear why a section at C-2 or C-3 would fail to relieve brachial plexus pain. If a complete section of the spinothalamic tract were made at C-1, above the first cervical root, one might hope that at best the upper level of analgesia would reach the skin level of C-5 or C-6. Such a section in the cervical cord, however, is fraught with too great danger to be recommended. Moreover, division of the spinothalamic tract even in the medulla as proposed by Schwartz and O'Leary[12] has fallen short of establishing a high enough level of analgesia to solve this problem. Adams and Munro[13] tried the operation on three patients (two unilateral and one bilateral) and correlated their results with histologic studies made at postmortem. They concluded that although the operation was not especially dangerous in skilled hands, it did not result in analgesia in the neck, shoulders or upper extremities. They believed, therefore, that the method was not superior to high cervical chordotomy *plus section of cervical roots*. Their medullary sections were made at the inferior pole of the olive or at the uppermost

radicle of the eleventh cranial nerve. The resulting level of analgesia did not reach above the skin level of D-2.

It was in relation to this particular problem that I became interested in that uniform discrepancy between the level of section and level of skin analgesia. Commensurate with the existing knowledge of anatomy, I could only attribute this overlap to the proposition that pain fibers progress cephalad in Lissauer's tract by four to six segments before their secondary neurones cross to take their place in the spinothalamic tract. It was, therefore, assumed that if one sectioned the spinothalamic tract by anterior chordotomy at D-1 and also sectioned the fibers in Lissauer's tract at the same level but on the opposite side of the cord, he should be rewarded by a level of analgesia up to the skin level of D-1.* The results of this procedure adequately confirmed the hypothesis and have been presented in a preliminary report.[14] Two attempts to apply this principle to a cervical chordotomy at C-3 met with discouraging results in that the presumed section of Lissauer's tract did not raise the level of analgesia to include the distribution of the brachial plexus. The opportunity for further work along this line has not presented itself.

After high cervical chordotomy respiratory embarrassment has appeared to be attributable to interference with function of the phrenic nerve and even more. There has been impairment of diaphragmatic breathing and use of the accessory muscles of respiration. Since the impairment has appeared more serious than that after phrenic exeresis, I believe that high cervical chordotomy may interfere with central integration of respiration. The author performed a unilateral cervical chordotomy at C-2 on a young woman suffering from intractable pain in the chest on one side, the pain being due to inoperable sarcoma. She located the pain as high as the supraclavicular fossa. The operation progressed quite smoothly and without complications in all respects but it was necessary to keep the patient in a Grinker respirator for the three months that she lived. Thus, aside from the hazardous nature of high cervical chordotomy, it fails to eliminate pain in the shoulder and upper extremity and hence the author believes that it has relatively little purpose. If there is indication to bring the level of effective analgesia as high as D-1 or D-2, he believes that a section in the medulla as described by Schwartz and O'Leary[12] and by Adams and Monro[13] is the procedure of choice by a wide margin. To eliminate pain in the shoulder and upper extremity, he believes that mesencephalic tractotomy such as that described by Walker[15] is a procedure which is thoroughly feasible and should be cultivated. He does not believe that high cervical chordotomy can be more than a chance taking operation to be followed by disappointing results. Few neurologic surgeons to the writer's knowledge have recommended this operation as having any value. Stookey[16] endorses it with surprising avidity but unfortunately does not discuss the level of analgesia attained nor does he cite a case that would support his claims. In the discussion of Stookey's paper, Penfield cited a patient on whom he did a unilateral anterior chordotomy at C-2 under local anesthesia. The patient died suddenly while the wound was being closed. Dr. Stookey replied that he had seen this before with bilateral chordotomies. To support the claims made for high cervical chordotomy, Stookey[17] refers to his own report of two cases of carcinoma of the breast in 1931. In one patient the chordotomy was unilateral at the second cervical segment and in the other it was bilateral at the second and third cervical segments. The report is short and the level of analgesia is not given. He refers also to the paper of Peet, Kahn and Allen.[18] These authors report the results of a bilateral cervical chordotomy on a young man with chronic infectious arthritis. The

* Testing skin analgesia for accuracy of level should be carried out laterally or on the back because the supraclavicular nerves from the cervical plexus maintain algesia over the upper anterior chest.

operation was as follows: *On the right*, the fourth and fifth posterior cervical roots were cut and a section in the cord made at C-8. *On the left*, the fourth posterior cervical root was cut and a section in the cord made at C-3. The cord sections were made 4 to 5 mm. deep and extended from the base of the dentate ligament only to the point of emergence of the anterior rootlets. In their photograph of the patient, a line is drawn just above the clavicle on the right and at about D-2 on the left. It is stated that pain and temperature sense was lost below these levels. This is a most unusual result, especially in view of the limited breadth of the chordotomy. Moreover, subjective relief and interpretations in a patient with chronic arthritis are not comparable to those in a patient with carcinoma pain. To abolish intractable brachial plexus pain it is necessary that the distribution be definitely within the field of analgesia. My purpose in this critical review is to express a strong conviction that high cervical chordotomy should not be misrepresented as a procedure lacking in serious hazards or as a procedure having any specific or special value. I have been unable to find an incontrovertible report of a case in which the level of analgesia definitely included the distribution of the brachial plexus after a high cervical chordotomy alone, nor have I been able to attain such a result myself. Foerster and others[18] have been lenient toward unilateral high cervical chordotomy while strongly opposing the bilateral operation because of a prohibitive mortality. In deference to these disadvantages, it would seem timely and appropriate to step above the high cervical danger zone and cut the tract at a medullary or mesencephalic level.

Spinothalamic Tractotomy, Spinal. This procedure has been variously referred to as section of the spinothalamic tract, anterior chordotomy, anterolateral chordotomy and plain chordotomy. As with many other procedures in medicine and surgery, a haphazard name has become so indelibly identified with this operation, that it is not likely that more descriptive and specific nomenclature can easily take its place. When spinothalamic tractotomy was first proposed and carried out by Spiller and Martin,[19] it was not anticipated that a field of surgery involving sections in the spinal cord, medulla and mesencephalon would so develop that even "anterior chordotomy" would become a too broad and inclusive term. Section of the descending spinal tract of the fifth cranial nerve, Sjöqvist's operation, has been referred to as "medullary tractotomy" or "intramedullary tractotomy." This term might be applicable to and easily confused with section of the spinothalamic tract at a medullary level. Hence, in view of the need for some standard nomenclature, I would like to suggest the following: That section of the spinothalamic tract be referred to as *spinothalamic tractotomy* to be followed or perhaps preceded by a term designating the location of the section, i.e., spinal, medullary or mesencephalic; that section of the descending spinal tract of the fifth cranial nerve be referred to as Sjöqvist's operation or as *trigeminal tractotomy*. Reference in this way to the descending tract as *the* trigeminal tract seems permissible.

Section of the spinothalamic tract on a spinal level has evolved to take its rightful place among standard recognized operations of definite indication and worth. It was necessary that considerable work be done, both experimental and clinical, in order that expectations and limitations, disadvantages and sequelae, optimum methods and location of performance could be allocated to the operative procedure.

It is not necessary to give a detailed account here of the results and the knowledge accrued from the work that has been done. The results of my own experience and researches are given in previous publications along with references to the works of others.[20–25] It will suffice to express my conviction that the D-1 cord segment (between the roots of C-8 and D-1) is the over-all optimum location for *spinal-spinothalamic tractotomy*, largely because the highest

spinal level of analgesia is obtained and the operator is more assured of accomplishing his objective. Evidence has been deduced[20] to show that the tract mediating pain and temperature resides more anteriorly in the cord than Spiller supposed, at least at the D-1 level. Hence, in order to obtain a complete and high level of analgesia the section should be carried well beyond the anterior rootlet line. The writer believes that inaccurate topographical localization of the spinothalamic tract was responsible for an era of misconception in respect to the physiology of the tract and the value of tractotomy.

Many intriguing questions have been aroused concerning function of the spinothalamic tract and experience with many cases of chordotomy has made it possible to throw a little light on its nature. Some of the conclusions are briefly as follows: (1) The sensibilities of pain and temperature have always been impaired together and remained undissociable in all the patients studied; (2) the spinothalamic tract appears to be the only tract that mediates the sensation of pain; (3) the author has encountered no findings that would support a hypothesis of bilateral representation; (4) there has been no evidence that sensibility to pain could regenerate or recur in the distribution of the severed fibers of the tract; (5) the fact that a knife may be passed through the tract in the conscious patient without pain is a fascinating revelation which at present adds another mystery to nervous integration; (6) at present it is reasonable to postulate that section of the spinothalamic tracts alone would result only in a loss of the sensibilities to pain and temperature, this being their specific function and *raison d'être*. The patient is aware of the deficiency only through appropriate stimulation and (7) the sensation of "itch" is undoubtedly a complex modality of pain and is completely abolished on section of the tract.

The untoward sequelae associated with spinothalamic tractotomy result from injuries to neighboring tracts which are, as yet, unavoidable. These complicating factors are minimal and often totally absent when the section is unilateral. They always appear to some degree when the section is bilateral. Those deserving prime consideration are: (1) Subjective and objective weakness of the lower extremities which is recoverable in a week to a month. One defect may remain indefinitely, a subjective weakness of the knees, probably due to impairment of the static, antigravity reflex integrated through the vestibulospinal tract;[23] (2) weakness of bowel and bladder sphincters requiring catheterization for one to two weeks; (3) loss of the possibility of orgasm in both male and female and (4) the writer has seen two patients who maintained a permanent and severe orthostatic hypotension after a bilateral operation.

As one might expect the debilitation accompanying cancer retards recovery and augments the disadvantages. The latter, however, by no means contraindicate the operation if pain is unbearable and otherwise intractable. While one should unequivocably always perform the unilateral operation if at all reasonable, it is equally as wise to do the bilateral operation if the migration of pain to the opposite side appears inevitable. Multiple stage operations in the management of a patient with intractable cancer pain soon makes the treatment as bad as the disease.

I do not believe that statistics in the form of percentages have any significance here. It is sufficient to report that the operative mortality from spinal chordotomy has been less than 1 per cent. The tenure on life has ranged from two months to five years and prognoses in this respect are notoriously inaccurate. Almost without exception the pain has been completely abolished or satisfactorily relieved. In the bilateral chordotomies some degree of motor weakness in the lower extremities and at least a transiently atonic bladder have resulted in every patient. The degree with which these deficits have occurred

correlates roughly with the debilitating effects of the cancer. It would appear to me that the comfort which the patient has during his limited time is the more significant objective and measure of success rather than the number of days or months of survival. It should be kept in mind that this discussion involves only the intractable pain due to cancer. The long term objectives and methods of treatment of pain from other causes are considerably different. The unwonted neurologic deficits are less prominent and when they appear after chordotomy, recovery from them is more rapid and substantial.

PREFRONTAL LEUCOTOMY

Although I have no personal experience in prefrontal leucotomy as a method of relieving intractable pain, I believe that its merits quite possibly may supersede those of the methods that have been described. Judging from the knowledge and experience that has and is being accrued from this operation and staking an abstract view anent the influence of prefrontal integration on the "intractability" of pain, it appears to be rational and promising. Prefrontal leucotomy looms as the third stage in the evolution of surgical methods in the treatment of pain. In the first stage, focus was upon the most immediate expediency and thus amputation and peripheral nerve section prevailed. In the second, an attempt is made, (i.e., by chordotomy) to interrupt specifically the pain-conducting system of fibers with otherwise minimal neurologic deficit. This leaves much to be desired, however, and may quite possibly prove to be transitional to a third stage in which the objective is to treat not only the unpleasant sensation of pain but also the psychic reaction to it and its sinister cause. The proposal is intriguing and provokes an endless chain of philosophic debates. Whether the wiggling of an injured earthworm is an expression of pain is an age-old controversy.[26] Whether decorticate man could suffer, will by nature of the problem always remain an abstraction.* But it is most reasonable to prophesy that if pain (in a broad sense with mental anguish and agony) can be reduced to a subconscious or unconscious reflex or projection, surgery will have reached the acme of achievement. This in a measure is the design of prefrontal leucotomy and the reports are encouraging.[28]

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SURGICAL TREATMENT OF EPILEPSY*

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MANY different pathologic conditions may be associated with convulsive seizures, so that an accurate diagnosis must be made in every case before any type of therapy, medical or surgical, can be rationally prescribed for the attacks. The etiologic factor may be suspected from the history but often cannot be demonstrated even after complete physical, neurologic, electroencephalographic, pneumoencephalographic and arteriographic examinations. In childhood, idiopathic epilepsy associated with a cerebral dysrhythmia is common but in adult life a convulsion is much more likely to be a symptom of a focal brain lesion.[63] Even if all the examinations mentioned fail to disclose a cause for the attack, the patient must still be suspected of harboring a small cerebral lesion which may later give more definite evidence of its presence.

With the exception of those cases of brain tumor, which require immediate surgery, the therapy of both idiopathic and symptomatic epilepsy is primarily medical. Only when anticonvulsive drugs in doses approaching the toxic levels have failed to control the attacks is surgical intervention justified.

IDIOPATHIC EPILEPSY

Trephination. Historical writers have produced evidence that the earliest operation for the relief of epilepsy was simple trephination. Numerous trephined skulls have been found in the ruins of ancient civilizations. This procedure was presumably done to release devils considered to be irritating the brain. Although the demonological conception has passed, the use of trephination in the treatment of epilepsy has not been abandoned entirely. Cutter[7] discusses five cases reported by Dudley in 1828 in which trephination and decompression was done. But in general, neurosurgeons concur with Penfield[37] who states that the results of subtemporal decompression do not justify the procedure in essential epilepsy.

Pneumoencephalography. Therapeutic effects of pneumoencephalography have been repeatedly reported since the introduction of this technic. In idiopathic epilepsy the procedure has not changed the course of the disease.[60] It is true that some patients have fewer attacks after the air injection but in most cases this improvement may be attributed to better medical management of the case. Thus Penfield[37] reports five of ninety-six patients to be seizure-free for an average of 2.8 years after pneumoencephalography. These patients were under sixteen years of age and had had attacks less than four years.

Freeing of Corticodural Adhesions. Ney[34] ascribes some cases of idiopathic epilepsy to local cortical tension due to traction by corticodural attachments at the vertex. Two hundred twenty-five patients with idiopathic epilepsy were operated upon and the adhesions relieved. The results were said to be good but the follow-up is not adequate to evaluate this procedure. Scarff[52] believes that there is a specific epileptic syndrome caused by anomalous pacchionian granulations extending into the motor area. Division of these granulations thus freeing the corticodural adhesions relieves many of the patients to some extent.

Miscellaneous Procedures. On the assumption that the sympathetic supply to the cerebral vessels is unstable in idiopathic epilepsy, sympathectomy has been performed. However, Penfield states, "Sym-

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pathectomy, however complete, does not make seizures impossible and is hardly justifiable as a treatment for epilepsy except in the presence of unusual evidence of abnormality of the sympathetic nervous system."[37] Removal of the carotid body and denervation of the carotid sinus have also been used in the treatment of epilepsy but Penfield[37] reports that the procedure is of no value unless specific abnormal hyperactivity of the carotid sinus exists.

With the purpose of preventing the convulsive seizure becoming generalized and thus preventing loss of consciousness in epileptics having a unilateral beginning of their attack, Van Wagenen[57] divided the corpus callosum. Six of ten patients so treated have had no generalized seizures since operation. Van Wagenen[58] has also made a hypothalamic puncture at the upper end of the pituitary stalk to induce dehydration by diabetes insipidus. In one case no convulsions had occurred at the time of the report.

In psychomotor epilepsy, negative spike foci have been found at the tip of the right or left or both temporal lobes.[17] Ablation of the tip of the temporal lobe and the focus has been carried out but the results are not yet known.

SYMPTOMATIC EPILEPSY

Many disorders of the brain are associated with convulsive seizures. The most common conditions include such congenital abnormalities as cerebral dysplasias (aplasia of cortex, microgyria, etc.) and vascular anomalies, degenerative states, inflammatory diseases, traumatic alterations of the brain and neoplasia. But not all patients suffering from these conditions have seizures. Probably less than 40 per cent are so afflicted. What factors render certain individuals susceptible? The complete answer to this riddle is unknown but some epileptogenic components are apparent.

1. *Location of the Lesion.* Cerebral lesions about the Rolandic area are prone to be associated with convulsions while polar lesions rarely have epileptic concomitants. In cerebral neoplasms and scars this factor has been studied in some detail.[2,15,19,20,29,35,41] List considers the histologic type of glioma to play an important rôle irrespective of the location of the tumor.[29]

11. *Severity of Cortical Damage.* Factors which tend to cause extensive cortical damage have been correlated with a high incidence of epilepsy. Thus penetrating dural wounds, infected wounds and extensive wounds are associated with convulsions about twice as often as a general, unselected group of head injuries. Retained foreign bodies, however, appear to have no relationship to the development of convulsions.[2,4,14,19,30,47,48,59,61]

111. *Heredity.* Is there an inherent predisposition to convulsions? The problem has been more adequately studied in post-traumatic epilepsy than other types of symptomatic epilepsy. Cobb gives statistics bearing on this question.[5] In 250 normal control subjects epilepsy occurred in 2.6 per thousand of 1,896 parents, siblings and offsprings. In 1,086 non-traumatic epileptics, epilepsy occurred in 21 per thousand of 9,139 relatives. In 235 patients with epilepsy who had suffered head trauma, there were 14 per thousand epileptic relatives. This figure is considerably higher than that occurring in the group of normal controls. The figures obtained in the group of post-traumatic epileptic patients studied at Cushing General Hospital differ from those of Cobb.[47] In this series only 4.5 per cent of the patient's relatives had convulsions compared to an incidence of 3.4 per cent in normal families and 17 per cent in epileptic families.[27] Ziskind[66] doubts that there is an inherited predisposition to epilepsy since it would have to be present in from 20 to 40 per cent of patients with head injuries and tumors. The epileptogenic factors are obviously not yet completely understood.

Type of Seizure in Symptomatic Epilepsy. There is nothing peculiar in the seizure of symptomatic epilepsy to distinguish it from that associated with idiopathic convulsions. In a series of cases, focal attacks are

much more common in symptomatic than in the idiopathic epilepsy.[2,14,47,61] The mode of onset of the seizure is usually an indication of the site of the lesion within the brain. Thus lesions in or near the motor area usually induce attacks beginning in the contralateral arm or hand, less commonly in the face and rarely in the leg. Sensory beginnings of the attack indicate the position of the lesion in the parietal, temporal or occipital lobes. The aura or onset of the attack is usually an excitatory phenomenon but paralytic manifestations are seen. Thus the most frequent aurae are twitching of the face, eyes, fingers, etc., but occasionally the onset is indicated by an inability to talk or a weakness or heaviness of the arm. Similarly in the sensory sphere, the aura may be excitatory or paralytic. For example, visual aurae may consist of a blurring of vision in one or both visual fields. This is frequently described as a black ball, dots, circular curtain moving across the visual field in an undulating or pulsating fashion. Frequently this scotoma begins in the periphery of a hemianoptic sector, proceeds across the vertical meridian to involve the normal field causing a complete loss of vision for a few minutes. With the onset of blindness a generalized attack sometimes develops. The visual aura may also consist of white or colored "lights" in the affected visual fields. When lateralized the hallucination occurs in the visual field contralateral to the cerebral wound.

Vertiginous aura manifested by dizziness just before a generalized attack is not an uncommon complaint and in some cases a true vertigo is present. Auditory aura and epigastric aura may occur at times. The epigastric aura of a feeling of uneasiness in the pit of the stomach, nausea, a choking sensation, cramping abdominal discomfort and vomiting are not so frequently encountered as in idiopathic epilepsy. Complex and bizarre aura may also occur. These may consist of combinations of auditory, visual and motor aura.

These focal manifestations may constitute the entire attack or they may progress to a generalized convulsive seizure. A true Jacksonian seizure is not often seen in symptomatic epilepsy nor are petit mal attacks as common as in the idiopathic seizures.[14,47,54,55,61]

ELECTROENCEPHALOGRAPHIC FINDINGS IN SYMPTOMATIC EPILEPSY

The electroencephalogram usually distinguishes the symptomatic from idiopathic epilepsies. It is based upon evidence of focal brain damage such as slow delta waves with superimposed sharp, spiky waves. This question has been most extensively studied in the post-traumatic epilepsies which will be discussed in some detail.

In a study of eighty-one cases of post-traumatic epilepsy Jasper and Penfield[23] found that in 90 per cent the electroencephalogram revealed a local area in one hemisphere from which random spikes or sharp waves were most prominent, usually upon a background of random delta waves. Slow waves alone, they believed, were not a reliable guide and positive electroencephalographic evidence of a focal lesion was present only when spikes or sharp waves were repeatedly recorded from the same discrete area.

In an early report Williams states that the character of the changes seen in the electroencephalographic record in traumatic epilepsy appear to be the same as in idiopathic epilepsy.[64] In a later report, however, he states that focal abnormality is found in a large proportion of cases of traumatic epilepsy in contrast to the group of idiopathic epileptics.[65] An abnormal electroencephalogram persisting after a head injury does not necessarily increase the likelihood of traumatic epilepsy; but if paroxysmal outbursts are present, the likelihood is increased by four times. If a patient's electroencephalogram shows larval epileptic outbursts, epilepsy is almost certain to supervene. Gibbs, Wegner and Gibbs[16] found that focal electroencephalographic abnormality is four times as

common in post-traumatic epileptics as in unselected epileptics and that the focal electroencephalographic abnormalities correlate with focal seizures. Focal seizure—discharges are twenty-one times as common among post-traumatic epileptics as among head injury patients without seizures. Roseman and Woodhall[49] reported paroxysmal electroencephalographic changes in fifteen cases of posttraumatic epilepsy; in twelve cases with focal seizures localized electroencephalographic discharges were present. In four of the fifteen patients with convulsions abnormal electroencephalographic foci were recognized first. However, there were another fifteen cases in the series with similar paroxysmal abnormal electroencephalographic changes but without clinical convulsions.

Kaufman and Walker[24] studied the electroencephalograms of 241 cases of post-traumatic epilepsy and of eighty-three cases of severe head injury but without seizures. The records were abnormal in 91.3 per cent of the epileptic and 77.1 per cent of the non-epileptic group. A focal abnormality was present in 83.8 per cent of the epileptic cases and in 67.5 per cent of the control group. The most common focal abnormality in both groups was the presence of slow waves, but this was much more frequent in the epileptic (73 per cent) than in the non-epileptic group (48.2 per cent). Paroxysmal abnormalities consisting of sharp waves or spikes which are usually considered indicative of epilepsy, occurred about equally (approximately 20 per cent) in the epileptic and control series. (Fig. 1.)

The epileptic character of a focus may be brought out by metrazol activation[25] or in sleep records.[18] Following a rapid intravenous injection of 200 mg. (2 cc.) of metrazol, within thirty to sixty seconds focal epileptic abnormalities usually appear in the electroencephalogram. This focal discharge consists of slow waves or humps having a frequency less than the alpha rhythm or single or multiple spikes, at times occurring rhythmically, giving the pattern of a localized electroencephalographic seizure. (Figs. 2 and 3.) This type of focal discharge occurs in about 67 per cent of a series of cases of post-traumatic epilepsy. If the procedure is repeated on another occasion, approximately one-half of the cases having no change on the first examination will have electroencephalographic alterations on the second trial. Abnormalities present before activation are usually aggravated by the metrazol. In about 88 per cent of the positive cases the focal electroencephalographic alterations induced by metrazol are paroxysmal. In 10 per cent of cases generalized electroencephalographic alterations consisting of single or multiple slow waves or spikes are present simultaneously in tracings made from several parts of both sides of the head, giving a pattern of a generalized electroencephalographic seizure.

If the metrazol activation is made after the patient has had no anticonvulsant medication for several days, clinical seizures similar to the patient's usual attack will occur in about 15 per cent of the cases. Clinical seizures have not occurred when anticonvulsant medication was not interrupted, but in a small percentage of cases the patient may experience a sensory or motor aura.

The epileptic manifestations may be brought out by spontaneous or induced sleep while the patient is having an electroencephalographic record made.[18]

EPILEPSY ASSOCIATED WITH BRAIN TUMOR

Convulsive attacks are a common symptom of brain tumor. Sargent[51] reported an incidence of convulsions of 30.9 per cent in a series of 270 patients. Parker[35] in a series of 313 patients with cerebral tumor found that sixty-seven (21.4 per cent) had major epileptic convulsions. Dowman and Smith[10] found that seizures occurred in 39 per cent of 100 cases of intracranial growth. In a statistical survey of Bailey's cases from the University of Chicago

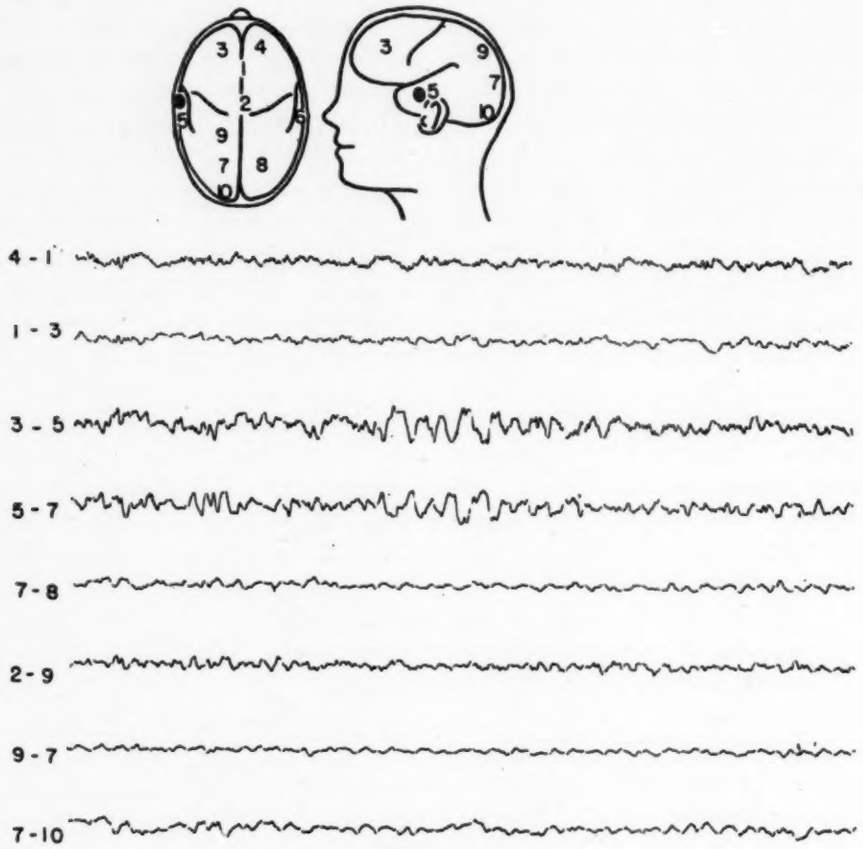

FIG. 1. Electroencephalogram of a patient suffering from post-traumatic epilepsy. The slow wave focus at the site of the small cranial wound just above the ear is evident. The leads are indicated on the traces. The horizontal line at the base represents an interval of one second and the vertical line a calibration of microvolts.

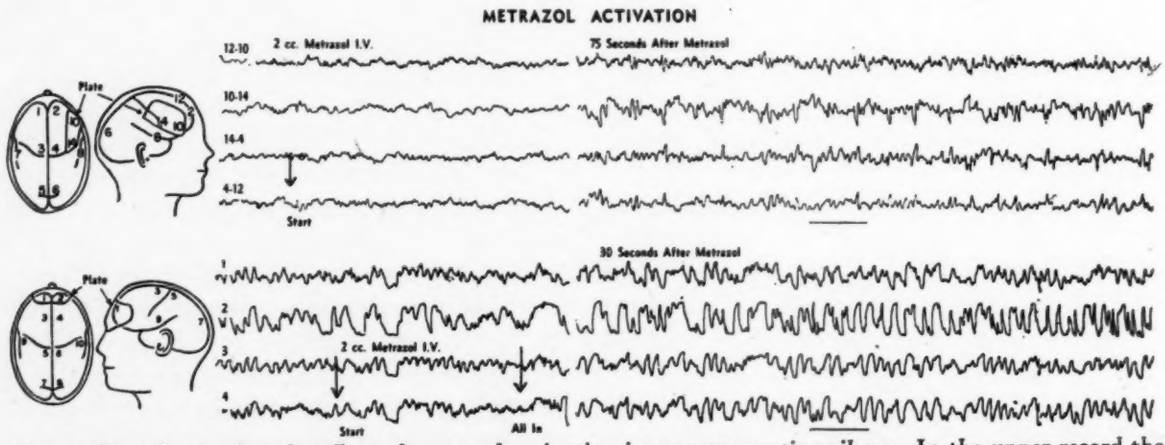

FIG. 2. Records showing the effect of metrazol activation in post-traumatic epilepsy. In the upper record the spiking at point 14 is apparent. In the lower record the marked slowing of the cortical rhythm with particular irregularities in the second trace is evident. An interval of one second is indicated by the horizontal line at the base.

Ley and Walker[28] found that 25.7 per cent of the patients suffering from tumor of the brain had generalized convulsions. Penfield et al.[41] report an incidence of 51 per cent in intracerebral and 62 per cent in extracerebral neoplasms.

the greater is the likelihood of convulsive seizures developing.[29,41] Gibbs[15] and Groff[20] have found that a tumor of the temporal, parietal or frontal lobe produces seizures more frequently than one in another region. Infratentorial and pituitary tumors

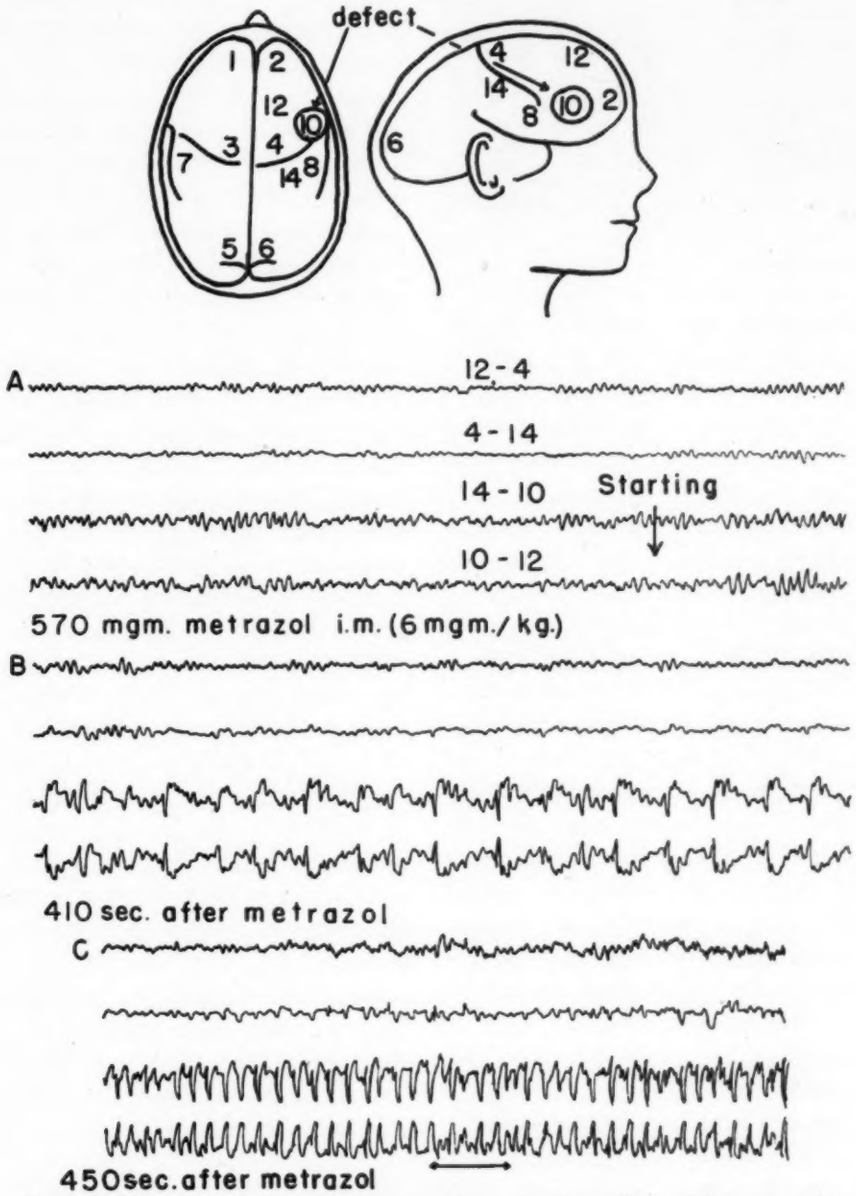

Fig. 3. Electroencephalogram to show the activating effect of intramuscular metrazol. The leads are as indicated. The spiking from point 10 is well shown in the lower two sets of tracings. An interval of one second is indicated by the horizontal line at the base.

The incidence of convulsive attacks in patients with cerebral tumor varies with the type and location of the neoplasm. The nearer the tumor is to the central sulcus rarely are associated with convulsions.[41] Tumors with a long life history are likely to be associated with convulsive seizures. In Penfield's series of cases[41] 37 per cent

FIG. 4. Angioma of left inferior frontal region inducing convulsive seizures.

of the glioblastomas had seizures, 70 per cent of the astrocytomas, 68 per cent of the meningeal tumors and 92 per cent of the oligodendrogliomas. List found a similar relationship between epilepsy and histologic type of tumor.[29]

Removal of the tumor frequently abolishes the attacks. This result may be enhanced by care at the time of craniotomy that no devitalized or macerated cerebral cortex is left at the site of the tumor. Penfield et al.[41] report that of twenty-nine epileptic patients with meningiomas, nineteen continued to have seizures after removal of the tumor. In a series of twenty-one non-epileptic patients with meningiomas nine had developed seizures after operation. In a group of twenty-seven epileptic patients with astrocytoma, twenty-three continued to have seizures; of six patients without preoperative seizures, three later developed seizures.

Following operation, a patient who has had convulsive attacks preoperatively should continue to take anticonvulsant medication. If he remains attack-free for a period of two to three years, the anticonvulsant medication can then be gradually discontinued. The question arises as to whether all patients with brain tumor should receive postoperative anticonvulsant medication since a certain percentage of them will develop seizures. This is a matter of individual choice but in most cases medication is not given until the patient has developed attacks.

EPILEPSY ASSOCIATED WITH CONGENITAL LESIONS

Congenital lesions of the cerebrum are numerous in type and degree. Epilepsy is an infrequent symptom of certain of the congenital lesions although in the localized aplasias and especially the vascular abnormalities it is particularly common. (Fig 4.) Usually included in the congenital abnormalities are cerebral lesions incident to delivery such as cerebral hemorrhage, contusion or laceration and vascular occlusion. These injuries cause the formation of atrophic gyri and cysts.

The treatment of epileptic seizures secondary to congenital lesions is primarily medical. With the use of appropriate doses of anticonvulsant medication many of the cases can be controlled. Roentgen therapy is of value in some cases of vascular abnormality. The indications for surgery in the treatment of epileptogenic congenital lesions are few even in many of the cases not controlled by medication. Frequently the damage to the cerebrum is diffuse and widespread so that little can be accomplished by surgical intervention. Surgical therapy is indicated in those cases not controlled by adequate anticonvulsant medication in which there is neurologic, roentgenologic or electroencephalographic evidence of a focal lesion. The congenital vascular abnormalities constitute a special problem. The former high mortality involved in their surgical treatment has been reduced by present methods of hemostasis and adequate blood replacement.[46]

EPILEPSY ASSOCIATED WITH INFLAMMATORY DISEASES

There are a number of types of inflammatory disease of the central nervous system which may result in convulsive seizures. The most common of these are the pyogenic infections. This includes the acute pyogenic meningitides, chronic arach-

noiditis secondary to a meningitis and pyogenic brain abscess. Seizures occurring in an acute meningitis are, like fever and delirium, only temporary symptoms of the disease. Only rarely will a chronic convulsive state develop secondary to pyogenic meningitic involvement. Seizures are common symptoms of an acute brain abscess and not infrequently continue when the abscess has been aspirated, drained or ablated. The convulsions may begin a number of years after drainage of the abscess.[1,6,21,23] Occasionally a chronic encapsulated abscess which was never surgically treated may remain for years unrecognized with symptomatic convulsive seizures. (Fig. 5.)

The convulsive seizures of neurosyphilis are well known. Tuberculomas are frequently diagnosed as cerebral tumors and may be associated with convulsive seizures. The yeasts and fungi may invade the central nervous system producing a chronic meningitis with occasional epileptic attacks. An amebic abscess is sometimes found within the cerebrum. Certain parasitic infections such as echinococcus, cysticercus and the shistosomes involve the cerebrum with convulsive seizures as a common symptom. These infections, becoming more common in this country, may be diagnosed in some cases by calcification in the multiple lesions.[8,9]

The treatment of the epileptic seizures in these various inflammatory diseases consists of specific therapy directed against the causative organism and anticonvulsive therapy.

If medication does not control the seizures, a surgical exploration and ablation of the epileptogenic zone is indicated.

EPILEPSY ASSOCIATED WITH CEREBRAL SCARS

Following a cerebral wound, a firm scar forms binding together the cerebral cortex and dura mater. In many cases a layer of blood in the subdural space becomes organized forming a membrane of varying thickness. At the time of operation for post-traumatic epilepsy this subdural membrane is found firmly adherent to the dura mater. It may be 2 to 10 mm. in thickness and quite extensive overlying as much as one-half of a hemisphere in some cases. The brain adjoining the meningocerebral scar in some cases appears normal, but frequently it is yellow or brown, softened and cystic. The arachnoid is often thickened and opaque, occasionally containing white calcified plaques. Because of atrophic gyri, arteries and veins usually concealed in sulci may present on the surface of the brain.[61] (Figs. 6 and 7.)

Histologically a meningocerebral cicatrix consists of connective tissue with collagen fibers and blood vessels. The cicatrix may contain neuroglia cells of the piloid variety but usually no ganglion cells.[38,42] Penfield has described the anastomosis of the vascular channels in the connective tissue with the vaso-astral network of the brain. Because of this anastomosis, as the cicatrix contracts there is a resultant pull and traction on the brain.[36] Between the scar and normal gray matter is an intermediate zone of partially destroyed gray matter in which there is evidence of progressive neuronal destruction in small patches surrounding blood vessels. The presence of compound granular corpuscles and changes in the neuroglia are said to be evidence of the progressive nature of the lesion.[43] This histologic picture is the same whether the patient does or does not develop epilepsy.[3,26,43]

The distortion of the brain due to the cerebral scarring is well illustrated in pneumoencephalograms. The most common pneumoencephalographic finding in the cases of penetrating head injuries is a bilateral ventricular enlargement with an outpouching at the site of the skull defect. In a series of 109 patients with post-traumatic epilepsy who had pneumoencephalograms made from three months to three years after their head injury, this type of pneumoencephalographic appearance was present in sixty-three cases. In

FIG. 5. A calcified capsule of a chronic abscess secondary to otitis media. A, lateral roentgenogram of the skull; B, extirpated abscess.

twelve cases there was unilateral ventricular enlargement with an outpouching and in eight cases a simple unilateral ventricular dilatation on the side of the lesion.[61] (Fig. 8.) The subarachnoid space, usually on the side of the lesion, is poorly demonstrated in many cases, but occasionally cysts or dilated sulci are visualized. The pneumoencephalograms of patients suffering from closed head injuries were either normal or had symmetrical ventricular dilatation. Of the entire group of 109 patients, only fourteen pneumoencephalograms were reported as normal. These ventriculographic changes are not peculiar to cases of post-traumatic epilepsy. Practically the same findings were present in a non-selected group of head injuries re-

ported by Troland et al.[56] although the incidence of severe ventricular distortion was higher in the epileptic series than in the unselected one. Convulsive attacks may occur immediately after a head injury or they may make their appearance many years later. For convenience most authors divide post-traumatic epilepsy into immediate (attacks in the first twelve to twenty-four hours), delayed, (attacks in first four weeks) and late epilepsy. Penfield[44] believes that the "early fit" should not be included in the discussion of post-traumatic epilepsy. It is true that a large percentage of patients with immediate attacks do not continue to have recurring seizures. In Penfield's group of 407 cases of civilian head injury there were fourteen patients with early seizures out of which only 4 or 28.5 per cent developed traumatic epilepsy.[44] In Wagstaffe's 377 cases of gunshot wounds of the head five of twelve patients with early seizures became chronic epileptics.[59] Ascroft[2] states that in about one-third of the patients having fits, the attacks are more or less of a transient character. Maltby[30] reported thirty-four cases of convulsive disorder following penetrating craniocerbral injury. In one-third of these cases the attacks occurred within a week or two of the injury; three of these patients continued to have seizures.

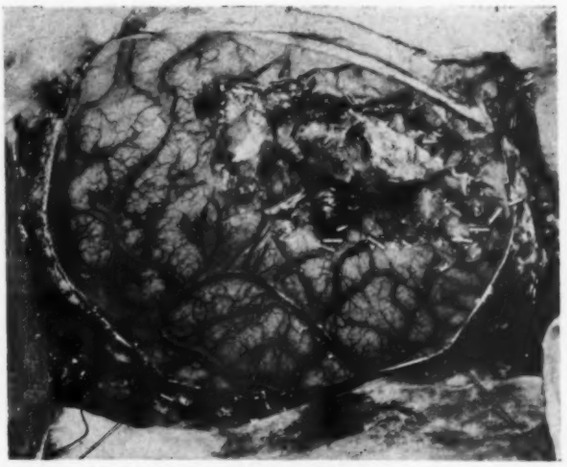

Fig. 6. Large meningocerebral cicatrix causing traction of the Sylvian vessels toward the scar.

In the majority of cases of post-traumatic epilepsy the attacks begin within the first few years. In a series of 500 cases of gunshot wounds of the head, Gliddon[19] states that 43 per cent of the cases appeared in the first year following injury. The average latent interval in twenty-six cases of post-traumatic epilepsy reported

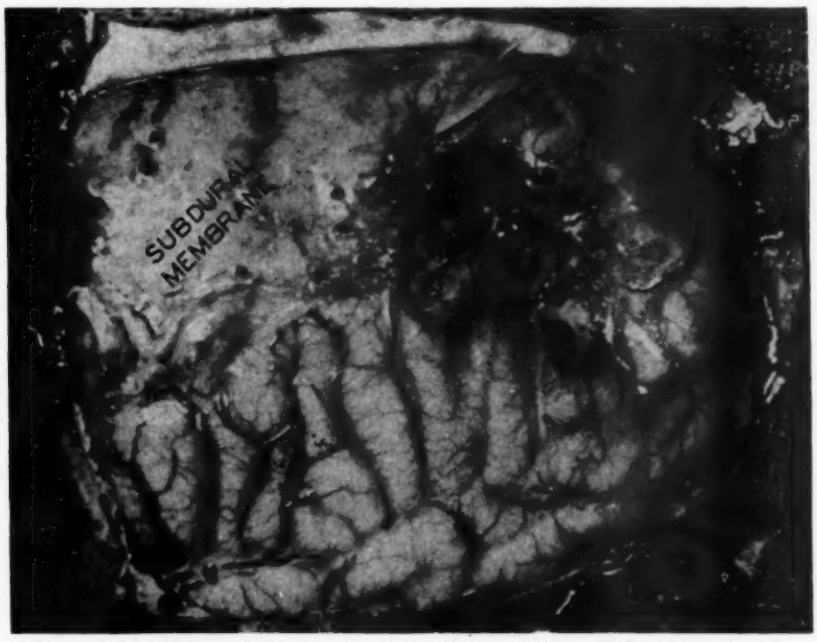

Fig. 7. Meningocerebral cicatrix with adjacent thin, subdural membrane.

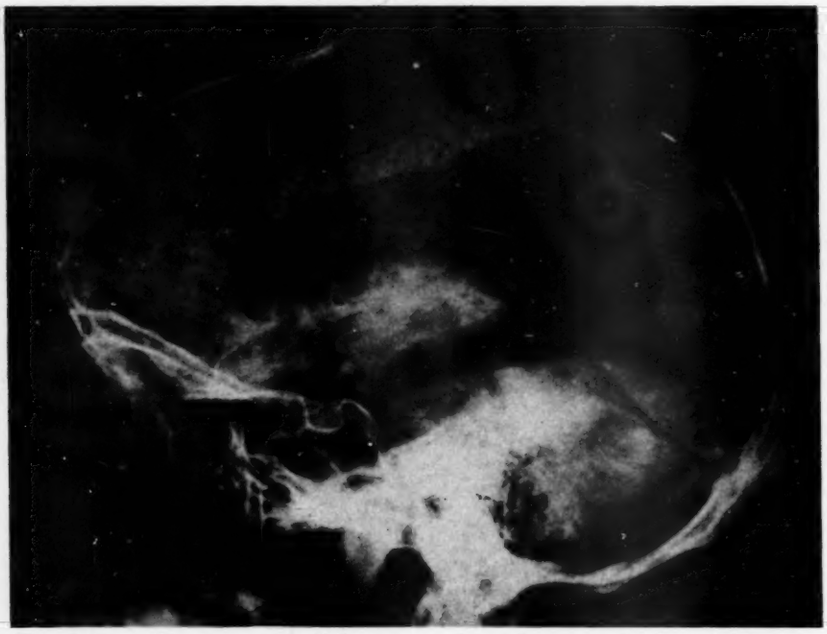

FIG. 8. Lateral pneumoencephalogram to show the marked trigonal dilatation and out-pouching in the direction of a skull defect in a patient suffering from post-traumatic epilepsy.

by Symonds[55] was two years, nine months. In seventeen cases it was three to eighteen months and in nine cases two to sixteen years. In another group of cases reported by Elvidge[11,12] the average time of appearance of attacks of forty cases of grand mal was 5.3 years. Twelve appeared within the first year, twelve more in one to five years and ten more in five to ten years. One case occurred twenty-five to thirty years after injury. The average time in sixteen cases of petit mal was 5.82 years. Ascroft[2] states that at the end of the second year the rate of onset reaches a very low figure and that only about one patient in twenty-seven developed epilepsy between the fifth and twentieth years. In a series of 246 cases of post-traumatic epilepsy treated at Cushing General Hospital, 27 per cent of the cases had the first seizure within three months and 30.7 per cent between three and six months after injury.[47]

The reported incidence of post-traumatic epilepsy varies considerably. This is probably due to several factors. There are differences in the type of head injury occurring in the various groups of cases. Criteria vary as to what to include as a convulsive seizure. Some figures include immediate attacks while others omit these. In a series of 500 cases of gunshot wound of the head including all cases having had one incident labelled epilepsy at any time following the head injury, Gliddon reports forty-nine cases of epilepsy, an incidence of 9.8 per cent.[19] In a similar series of 377 cases with the time of onset varying from two months to seven and one-half years, Wagstaffe[59] gives the same incidence, 9.8 per cent. In other reports of series of cases of gunshot wounds of the head, the incidence of convulsive seizures is as follows: Rawling,[48] 452 cases, 22 per cent; Stevenson,[54] 17,300 cases, 1.5 per cent; Sargent,[51] 18,000 cases, 4.5 per cent; Cushing,[4] 34 cases, 32 per cent; Maltby,[30] 200 cases, 17 per cent. In a series of 407 cases of civilian head injury, Penfield[44] found an incidence of epilepsy of 2.7 per cent. From these figures it can be seen that the incidence of post-traumatic epilepsy including all types of head injury varies from 1.5 to 34 per cent.

SURGICAL TREATMENT OF SYMPTOMATIC EPILEPSY

When medical management fails to control convulsive seizures due to a cerebral lesion, surgical procedures may offer relief if no contraindications exist. It is not wise to subject individuals who are aphasic, paraplegic or quadriplegic and confined to bed, to a serious operation for the relief of convulsive seizures which play only a minor rôle in their disability. If a patient is mentally disturbed or impaired to such a degree as to render him or her incompetent of following medical advice, the advisability of operation is doubtful. Because a cortical resection constitutes a major surgical procedure, the general condition of the patient should be as good as possible prior to operation. If the primary wound was infected or did not heal by primary intention, it is well to delay cortical exploration for at least six months to a year after the wound has healed. When the operative procedure is then done, chemotherapeutic agents, penicillin and sulfadiazine should be used pre- and postoperatively.

Just as in the cases of idiopathic epilepsy, a number of surgical procedures have been tried in the treatment of symptomatic epilepsy. Meredith[32] has described an operation for internal communicating hydrocephalus following certain traumatic or inflammatory lesions associated with seizures. The operation consists of a transcortical incision through a relatively unimportant portion of the less dominant cerebral hemisphere leaving a large stoma into the lateral ventricle. At the same time the choroid plexus is removed from the opened ventricle. Siris[52] reported five cases of focal epilepsy treated by encephalomyopexy. Three patients had not had seizures for five to seven months postoperatively and the other two had diminished frequency and intensity. Marsh[31] stated improvement occurred in some cases of traumatic epilepsy in a series which had been given radiation therapy. It is difficult to evaluate these different procedures because of the small number of cases reported.

The usual procedure used for the treatment of post-traumatic epilepsy is the surgical removal of the epileptogenic focus. Until recently it was thought that the lesion, scar, tumor, softening, etc., was the focus responsible for the seizures, but with the use of electrocorticography it has now been clearly shown that the marginal cortex is the source of epileptic discharges. In a recent discussion of post-traumatic epilepsy Penfield[45] stated: "We must identify more definitely, not the gross lesion but the actual focus of neuronal hyperirritability. It is the focus in which electrographic spikes and sharp waves originate and which causes the attacks."

The question arises if the epileptogenic focus is stable and if it does not with the passage of time set up other epileptogenic foci? These questions cannot be answered with assurance at this time although the available evidence favors the stability and constancy of an epileptogenic focus. Whether secondary foci will form if the primary lesion is left, remains for further research. Perhaps that is the explanation for certain failures.

Cases suitable for this type of surgical procedure must have some evidence of localization of the origin of the seizure. These may be apparent from the history of the attack, observation of the seizure, neurologic findings, electroencephalographic examination either spontaneous or activated, or roentgenograms of the skull with or without air injection. The localization so determined will indicate the proper placement of the osteoplastic flap.

Anesthesia. Local infiltration of the scalp along the proposed incision is the anesthesia of choice, because it does not interfere with the cortical activity nor the state of consciousness of the patient who can report subjective experiences during the cortical exploration. Morphine may be used to allay restlessness or anxiety.

Operative Technic. A large osteoplastic flap is turned down to expose the epilepto-

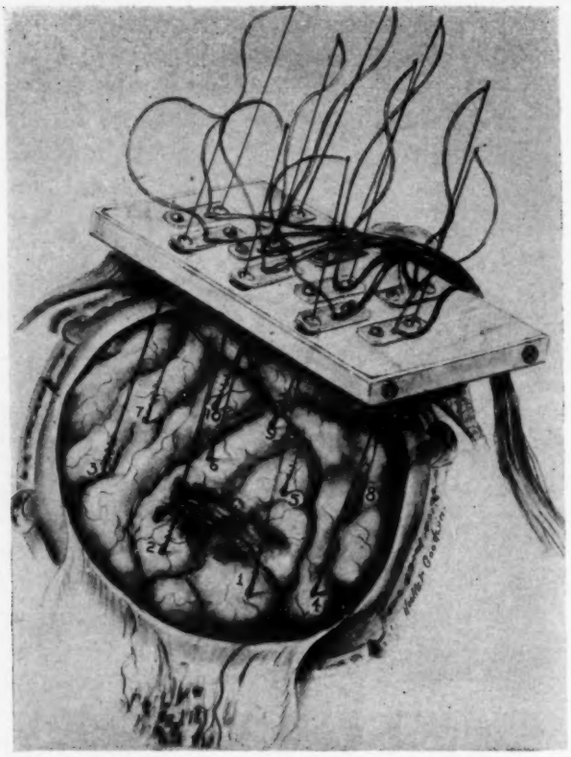

Fig. 9. Sketch to show the method of application of cortical electrodes.

genic focus. Occasionally the type of skin incision may be dictated by previous scalp wounds. A tantalum plate may have to be removed. In most cases the entire procedure can be done in one stage. In certain instances, however, where the lesion lies close to the midline necessitating exposure of the longitudinal sinus it may be wise to perform the operation in two stages. In the first stage the bone flap is turned down and tantalum foil or plate placed over the longitudinal sinus. This will decrease the likelihood of serious bleeding should the patient have a seizure as the result of cortical stimulation.

The dura mater must be reflected carefully to avoid tearing subdural adhesions. Sharp dissection is advisable to cut meningocerebral scars. If there is an extensive adhesion, it may be better to open the dura mater only at the margin of the lesion where the epileptogenic focus is thought to lie.

When the lesion and surrounding cortex have been exposed, the cortex is systematically explored by electrocorticography and electrical stimulation.

A special electrode holder* is clamped to the margin of the bone. Ten electrodes are placed on the exposed cortex to record the spontaneous and induced activity. (Fig. 9.)

Penfield and Jasper[40] describe the localized electrocorticographic alterations in focal epilepsy as consisting of large spikes or sharp waves appearing in random sequence against a background of lower amplitude activity arising from the border zone between the lesion and normal cortex. In fourteen of thirty-nine cases examined by electrocorticography Walker et al.[62] found such focal spontaneous epileptic activity confined to a portion of one gyrus. (Fig. 10.) Occasionally such a

*The electrode holder used in this Clinic was designed and made by Dr. C. Marshall.

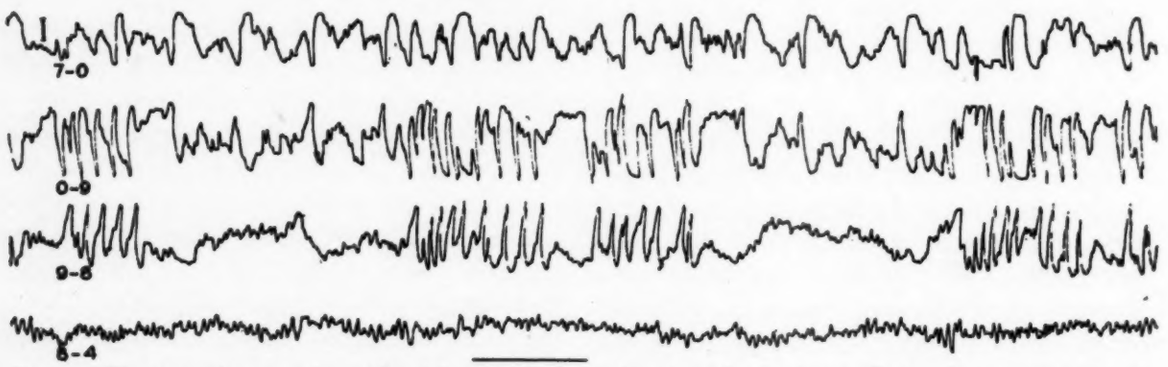

Fig. 10. Electrocorticogram from the right inferior frontal region of a patient suffering from post-traumatic epilepsy, the result of a severe penetrating wound of the head. The spiking activity at both points 0 and 9 is well shown. The horizontal line at the base indicates an interval of one second.

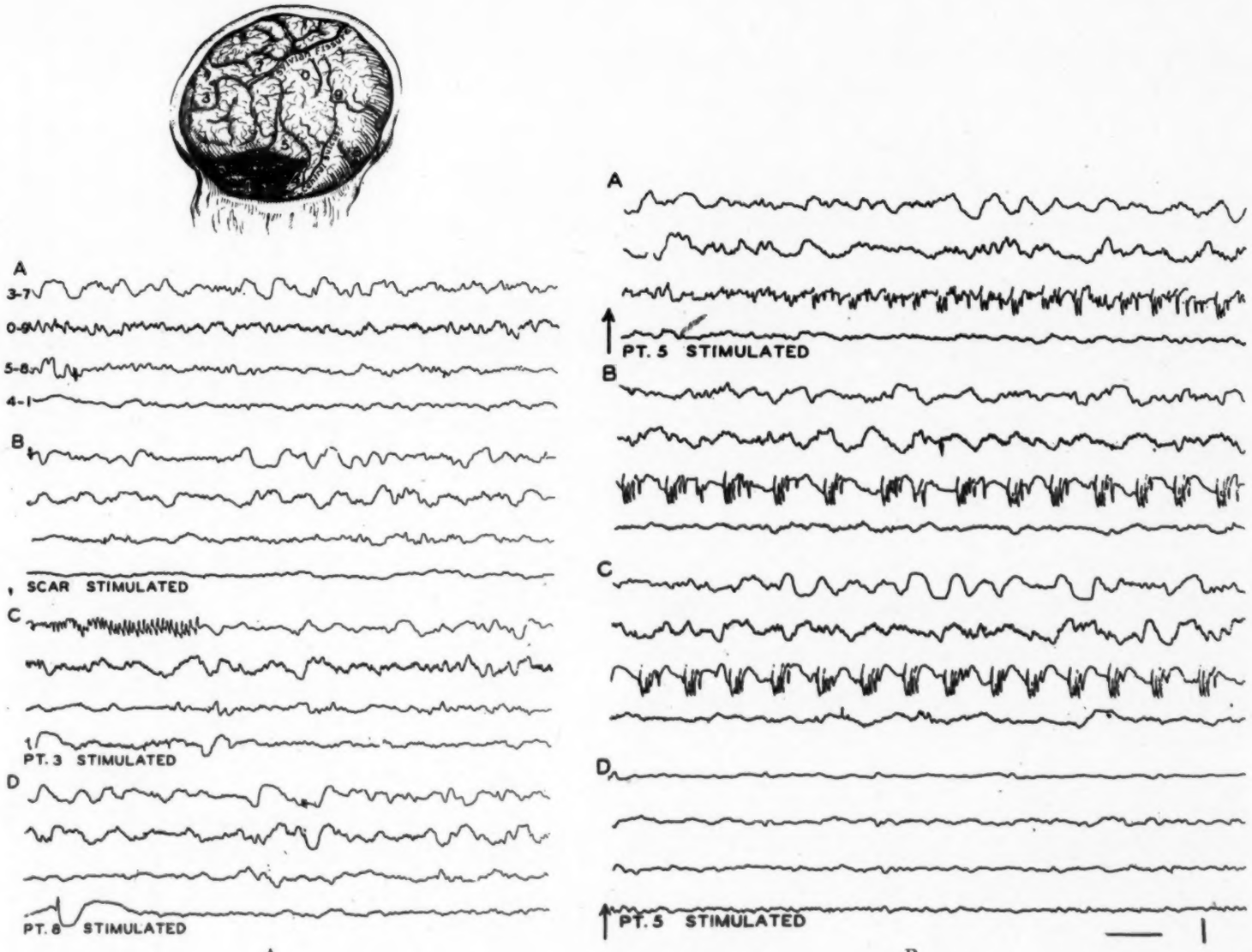

FIG. 11. A, electrocorticograms showing the technic of electroactivation. The electrodes are placed as indicated on the diagram. Stimulation with a 2.0 volt 60 cycle sine wave current for five seconds at the points indicated caused little alteration. B, stimulation of point 5 induced long-lasting localized after-discharge seen in A, B and C traces taken at one-minute intervals. After removal of the cortex of the triangular gyrus beneath point 5, stimulation no longer caused an after-discharge. The horizontal line at the base indicates an interval of one second and the vertical line a calibration of 50 microvolts.

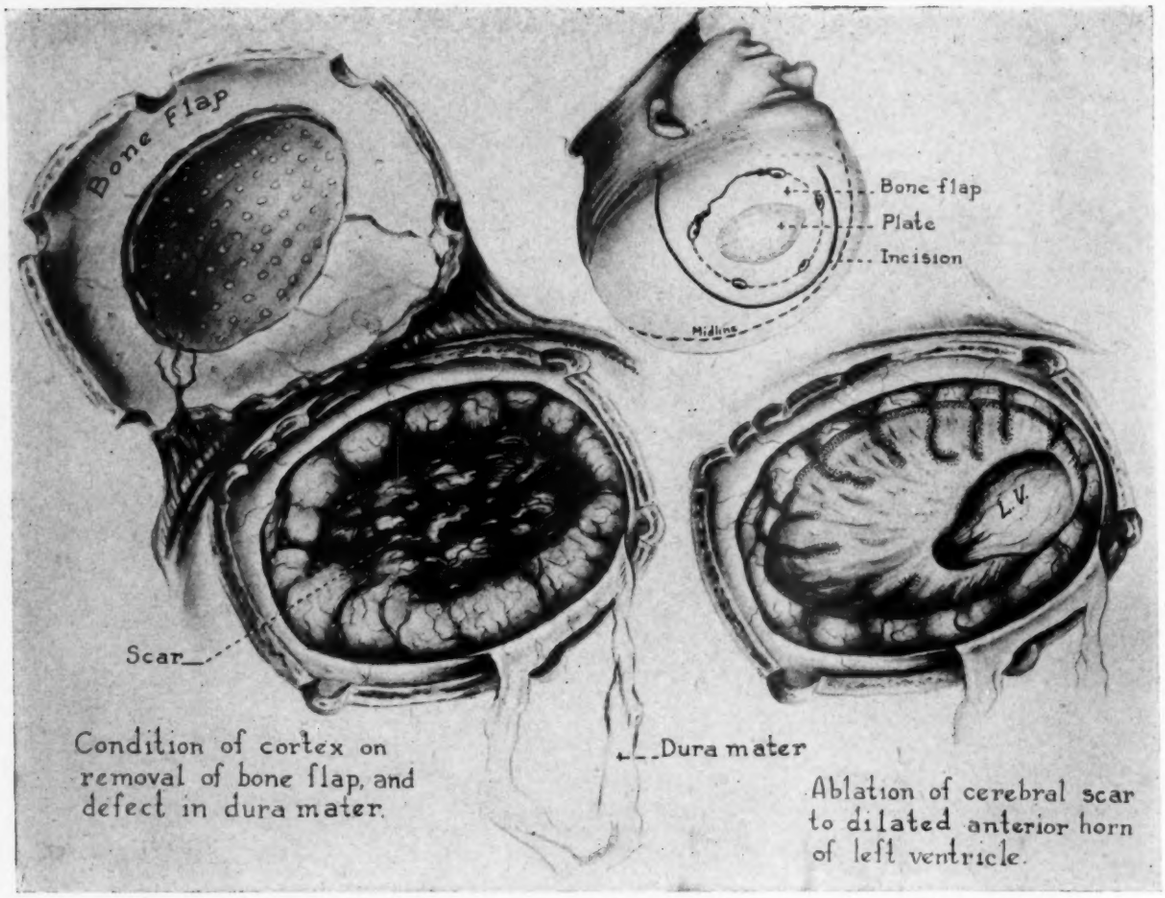

FIG. 12. Sketch to illustrate the surgical removal of a meningocerebral scar to the ventricle.

focus was found in normal appearing convolutions some distance from the scar.

The cerebral cortex is then explored using a sine wave current from 1.5 to 2.5 volts for five seconds. The electrical activity of the cerebral cortex, the motor responses and the patient's subjective sensation at the time of stimulation are noted. If no change is observed in the electrocorticogram after a minute interval, another point is stimulated and so on until the entire cortex has been mapped. An after-discharge lasting for a few seconds at the point stimulated is considered within physiologic limits. However, generally an area of cortex adjacent to the scar can be found which on stimulation induces a long lasting spiky after-discharge. This epileptic discharge has been seen to persist for as long as twenty-eight minutes. (Fig. 11.) In many cases the sensory aura which precede the patient's spontaneous attacks occur with this after-discharge. Occasionally the electrocorticographic attack progresses to a clinical seizure. But in some cases the individual does not have any motor or sensory concomitants during the electrocorticographic discharge.

With this technic the likelihood of the patient developing a severe seizure is much less than with the use of uncontrolled electrical stimulation. There is evidence to suggest that the focus found by electro-activated electrocorticography and that localized by electrical stimulation alone are not quite identical. On many occasions the after-discharge is present for several minutes and changing in character before the patient notices the aura of his attack. Frequently the latter is associated with decreased electrocorticographic activity. This suggests that the

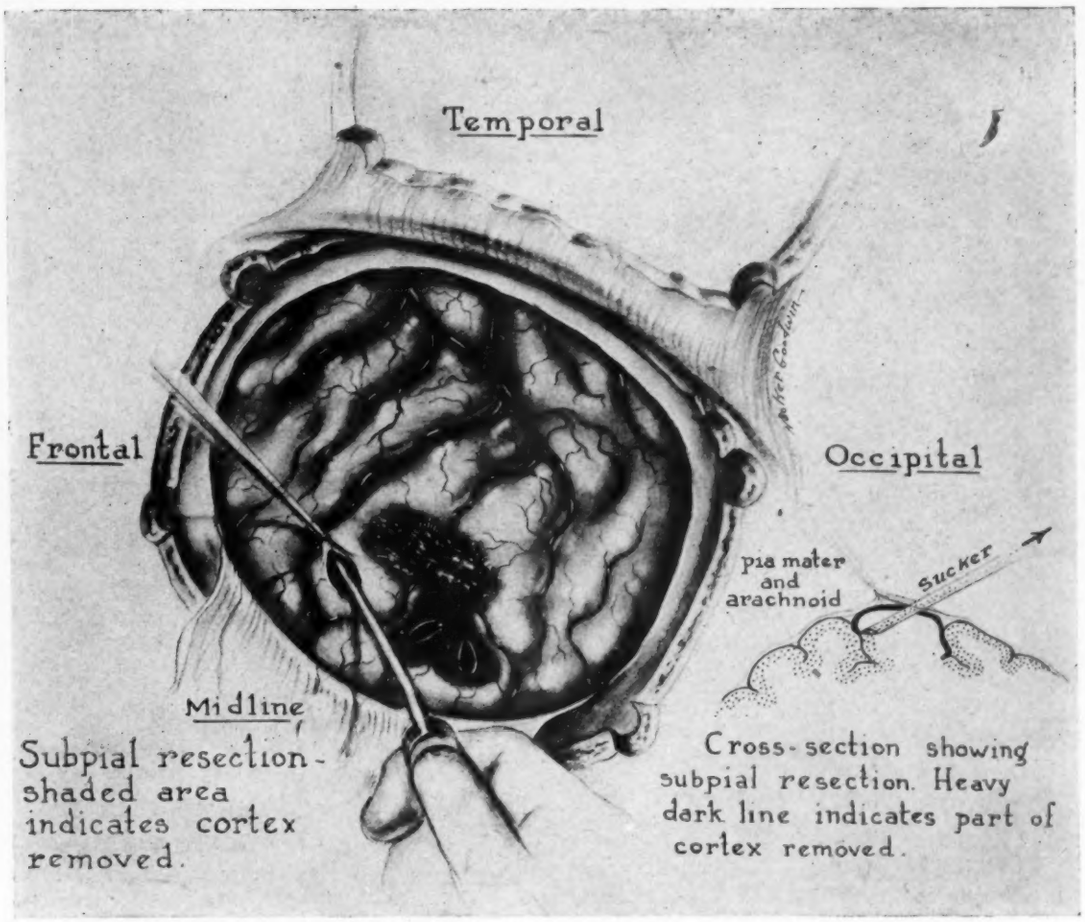

Fig. 13. Sketch to show the technic of subpial resection of an epileptogenic focus adjacent to a meningocerebral scar.

electrocorticographic focus is a primary focus which fires a secondary focus initiating the aura of the attack.

By rearrangement of the cortical electrodes and push-pull recording the epileptogenic focus can be delimited. It is usually adjacent to the cerebral scar but occasionally in a gyrus slightly removed. Multiple foci are occasionally present and a focus may migrate rarely from one cortical area to another. In most instances, however, the focus is constant and the typical after-discharge can be reproduced time after time.

In those cases in which the clinical aura and electrocorticographic manifestations of an attack are induced it seems probable that the stimulated focus is responsible for the patient's spontaneous attacks. In those cases in which only the electrocorticographic epileptic manifestations can be elicited, the evidence that the point stimulated is the origin of the attacks is not conclusive, even though there is no doubt that such areas are functioning abnormally and are potentially if not actually epileptogenic.

The epileptic focus having been located and its extent determined, it is removed by one of several technics, excision of the focus and the scar to the ventricle, removal of the focus and the scar to normal white matter or subpial resection of the focus.[13,22,50] Subpial resection of the focus is done by making an incision in an avascular portion of the pia arachnoid and ablating the cortex by subpial dissection and suction. The involved gyrus is removed completely from trough to trough. (Figs. 12, 13 and 14.)

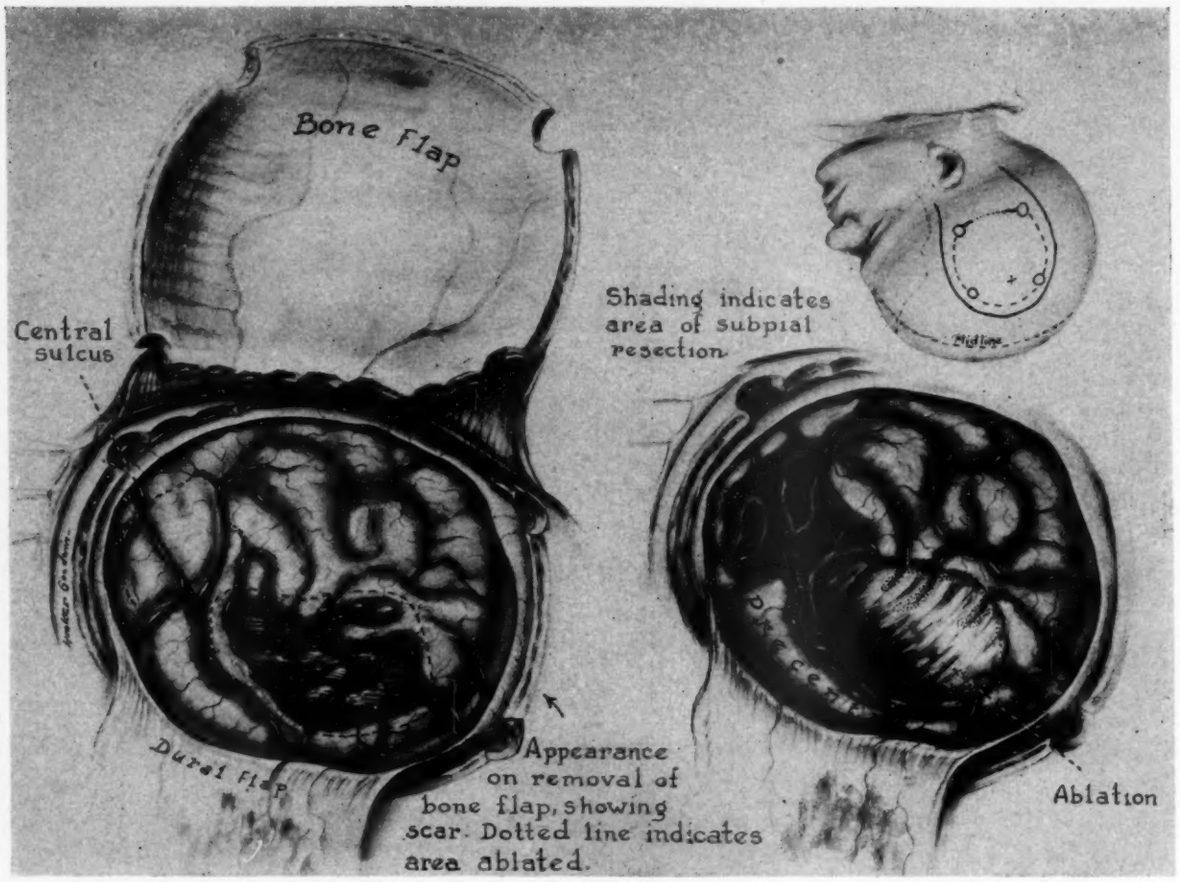
Bone flap
Central sulcus
Shading indicates area of subpial resection
Dural flap
Appearance on removal of bone flap, showing scar. Dotted line indicates area ablated.
Ablation

FIG. 14.

The decision as to which of the several technics to use will depend on evaluation of the individual case. It is not desirable to leave the patient with a more severe neurologic deficit than he had previously. If the scar is large and adjacent to the motor area, extensive resection may aggravate a paralysis or aphasia. The danger of complications is lessened if the ventricle is not opened. In the occasional case in which a clear cut focus cannot be demonstrated, it is probably well to excise the scar and entire surrounding epileptogenic zone. Irrespective of which procedure is carried out, the blood supply to the remaining cerebral cortex is disturbed as little as possible.

Following removal of the focus the electrodes may be placed upon the adjacent cortex to determine any remaining epileptogenic tissue. In some cases before all convulsive activity can be eliminated a rather extensive subpial dissection of several gyri running into the scar must be carried out. During the ablation, speech and motor power are checked frequently.

Postoperative Medical Therapy. After any operative procedure for the relief of epilepsy the medical management, especially anticonvulsant drug therapy, must be continued for a period of several years. If no further attacks occur, the medication may be decreased at the end of two years and gradually eliminated over another two-year period. If seizures occur, the medication may be augmented or changed. The patient's social and economic rehabilitation will be greatly facilitated if, through vocational guidance, an occupation, suitable to his or her physical, mental and psychologic status, can be found. Continued guidance may be necessary to effect a satisfactory adjustment to all of these factors.

Results. The largest series of patients treated by surgical extirpations of focal epileptogenic lesions has been reported by Penfield and Erickson.[39] During a ten-year period 165 cases were operated upon with a mortality of 4.2 per cent. In the group of patients with excision of a meningocerebral cicatrix, 22.5 per cent were completely free from attacks, 64.5 per cent were at least 50 per cent improved, 11 per cent unchanged and 2 per cent worse. In the group of patients with a cerebral cicatrix, 19 per cent were completely free from attacks, 49 per cent were improved, 30 per cent unchanged and 2 per cent worse. In a group of 25 patients having negative explorations, 2.5 per cent were completely free from attacks, 22.5 per cent improved, 69 per cent unchanged and 6 per cent worse. At the present time it would seem that cortical excisions may offer help to a certain group of patients not benefitted by medical management.

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FRACTURES AND DISLOCATIONS OF THE SPINE*

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ONE of the most difficult and perplexing problems confronting a physician is the management of a patient with a spinal injury. This is particularly true when the spinal cord is injured or endangered. Not only does it involve the immediate treatment of a potentially dangerous fracture but also the care of a profoundly disturbed physiology. So altered are body mechanisms, it is only by constant effort and exchange of professional ideas that optimum anatomic and physiologic restitution is possible.

Spinal injuries are caused by direct or indirect trauma. Direct injuries result from a heavy object striking the spine, a crushing force applied to the spine or penetration by high velocity missiles. The more common vertebral injuries result from indirect trauma as caused by falls or diving accidents in which strong force is applied along the axis of the spine crushing or dislocating vertebrae.

Dependent upon the intensity, duration and direction of the force applied to the spinal column, extreme flexion, extension or rotation results in ligamentous tears, fractures or dislocations of vertebrae. Probably the fracture most frequently encountered results from falls or automobile accidents in which the spine is abruptly flexed by a tremendous force. Less commonly, fractures due to extreme extension of the spine occur. Although fractures with or without dislocation may occur at any level, they favor the levels of greatest vertebral mobility—the lower cervical region (C_5, C_6 and C_7) and the thoracolumbar junction (D_{12} and L_1). Aside from actual bone displacement, tearing of the vertebral ligaments or cartilages with their impingement upon the spinal cord or nerve roots may result.

Whatever the mechanism of indirect injuries, the resulting deformities of the vertebral canal are remarkably constant:

1. The vertebral canal is narrowed in its anterior posterior diameter by dislocation of a vertebra with or without fracture of the body, pedicles or laminae. Most frequently this type injury is encountered in the low cervical region where spinal mobility is greatest. It is with fracture dislocations that the most severe cord injuries occur. Conversely there may be considerable narrowing of the canal in the cervical spine without cord injury for in this region the canal is large.

2. Crushing injuries of the vertebrae with wedging of the bodies and posterior displacement of fragments into the vertebral canal are commonly seen in the thoracic region. Any portion of the vertebral arch, however, may be fractured and displaced to impinge upon the cord.

3. Complete obliteration of the spinal canal is found in some of the more serious cases. This is not uncommon in extreme fracture-dislocations of the twelfth dorsal and first lumbar vertebrae due to violence to the shoulders.

4. Depending upon the severity of trauma, the degree of flexion, extension or torsion of the spine, a variety of vertebral fractures may take place with a varying degree of bony or ligamentous encroachment upon the vertebral canal.

In any series of spinal cord injuries, a variety of neurologic disturbances is manifest depending upon the actual amount of cord damage. Edema of the cord may develop at the site of injury and extend

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several segments above and below. This may be sufficient to interrupt additional cord function for a short period. As it abates, function returns, at least in part, except in those cases showing evidence of a complete transverse lesion.

The impact of displaced bone upon the cord frequently produces diffuse hemorrhages as well as perivascular extravasations particularly in the gray matter. At times, collections of blood may occur in the subarachnoid, subdural or extradural space. With severe fracture-dislocations, laceration and even masceration of the cord is common.

CLINICAL PICTURE OF CORD LESIONS

Complete Transverse. Immediately following a transverse cord lesion from severe contusion or laceration, there is complete motor, sensory and reflex loss below the level of the lesion. If spinal roots above the site of bone injury are involved, further sensory and motor loss are evident. The urinary bladder distends and unless relieved by decompression, incontinence results. Intestinal peristalsis is diminished and obstipation is present. All voluntary bowel control is lost. Both pilomotor and sudomotor activity below the level of the lesion are lost. However, these functions often return to an exaggerated degree within a few weeks. Trophic skin changes may readily appear over bony prominences unless these parts are well protected. It is not unusual to see bullae over the sacrum and buttocks within a few hours after a complete cord lesion. Edema of the lower extremities and back is common. Depending upon the level of the lesion, numerous clinical pictures may result.

Lesions involving the upper four cervical segments produce quadriplegia with a sensory loss extending up to the clavicles. Paralysis of the diaphragm results from lesions at C_3 or above and breathing is possible only through the use of the accessory muscles of respiration. As in all cord lesions above the first thoracic vertebra, miosis from paralysis of the dilator of the pupil may be seen. Hyperthermia, the etiology of which has not been completely explained, is common. Mucus accumulates in the respiratory passages and aspiration is necessary. These are grave lesions and it is seldom that the patient survives for more than twenty-four to forty-eight hours. With complete cord transsections in the lower cervical spine, there is paralysis of the lower extremities and weakness of the upper extremities depending upon the level of the cord lesion. The hand grips are always weakened. Breathing is abdominal in type as the intercostal muscles are paralyzed. The sensory level is at the third or fourth ribs anteriorly and sensation in all forms is abolished below this level.

Crushing fractures of the thoracic spine (below the first thoracic vertebra) in peacetime usually result from severe flexion or extension injuries. The already small vertebral canal is further diminished in size by displaced fragments or by dislocation of vertebrae. The upper extremities are unaffected while all sensation and motor power is lost below the level of injury. With fractures of the lower thoracic and first lumbar vertebrae because of the close proximity of cord segments, it is rare to have but one segment involved. More frequently, several segments of the cord and portions of the cauda equina are crushed. By determining the level of sensation, motor power and reflexes, the upper level of cord injury may be ascertained.

With fractures below the first lumbar vertebra, it is the cauda equina which may be involved. As the cauda equina is made up of peripheral nerves, the prognosis is much more favorable than with injuries to the spinal cord. The extent of nerve injury is evident by the motor and sensory loss in the lower extremities.

Incomplete. With injury to but a portion of the grey or white matter of the cord at any one level, a variety of neurologic disturbances dependent upon the extent and severity of cord trauma occur. When in the cervical region, a partial to

complete paralysis of the upper and lower extremities on one side and loss of pain and temperature sensation of the contralateral extremities may be seen (Brown-Sequard Syndrome). The reflexes are inconstant, being diminished or absent in the paralyzed extremities. Another common clinical picture is a partial to complete bilateral paralysis of the upper and lower extremities, dependent upon the level of injury with preservation of some appreciation of external stimuli applied to the paralyzed area. Reflexes may or may not be elicited. Incomplete cord lesions in the thoracic region (below D-1) may present similar pictures except the upper extremities are uninvolved and the transition from absent or diminished to normal sensation occurs from the inguinal ligaments to about the third or fourth ribs.

Incomplete lesions of the lumbar cord (L-1) produce motor and sensory impairment limited to the lower extremities together with sphincteric and visceral disturbances which accompany almost all severe injuries of the spinal cord and cauda equina. There is partial to complete paralysis of one or both lower extremities. Knee and ankle jerks are usually absent. Anesthesia of the stocking type may be found associated with the more severe injuries.

Incomplete lesions of the cauda equina result in paresis to paralysis of individual muscle groups depending upon the number of nerve roots involved. Sensation is impaired or lost in the dermatomes supplied by these nerves. Since motor components of the cauda equina have power of regeneration, the prognosis for at least some recovery is much more favorable than in cases of spinal cord injury.

TREATMENT

The immediate treatment of a patient with or suspected of having a fracture of the spine with or without neural involvement is his careful transportation to a hospital, due caution being exercised against any movement of the spine. Too frequently these patients are picked up at the scene of accident and rushed to a hospital in the first available conveyance, no thought being given to the possibility of a spinal injury. Unfortunately, some patients have also been rendered unconscious and the possibility of cord injury is not suggested. Perhaps the best method of transporting any patient with a possible vertebral fracture is on a board or similar flat, unbending surface. As in any injury, all available detail regarding the production of the injury and especially the rapidity of onset of neurologic disturbance should be obtained. When first seen, many patients are in shock which is treated by the usual methods. Further treatment should be guided by the results of: (1) neurological examination, (2) x-ray studies of the spine and (3) lumbar puncture.

Neurologic examination should be carried out in detail and recorded for future reference. This is especially helpful when incomplete lesions of the spinal cord or cauda equina exist, for then any progression or recession of signs may be followed. Much, too, can be learned of the prognosis, for with a history of sudden onset of paralysis following injury with neurologic evidence of a complete physiological transsection of the cord, there is little or no hope for functional improvement especially if none is evident after a few days. In general, the more cephalad the neural injury, the more serious the injury and higher the mortality. In transverse cervical cord lesions, considerable respiratory dysfunction results and even with the best of care a large percentage of these patients succumb within ten days.

X-ray studies of the spine should be conducted with great care to avoid undue motion or handling of the patient. With suitable films, the extent of vertebral damage, displacement of bone fragments and their encroachment on the spinal cord can be ascertained. Lateral x-ray views of the spine are the most valuable views. These studies are extremely helpful in estimating neural damage, guiding treat-

ment and evaluating its results. At times, no x-ray evidence of fracture is found and spinal cord injury is believed to be due to vertebral luxation with recoil to its original position or to massive extrusion of a nucleus pulposus. This is not an infrequent finding in the cervical region. Fractures and fracture-dislocations of the sixth and seventh vertebrae are frequently overlooked due to technical difficulties in obtaining suitable delineation of the lowermost part of the cervical spine.

Lumbar puncture is carried out as soon as the condition of the patient permits, due care being taken not to disturb the spine. The presence of bloody spinal fluid usually indicates a severe injury of the spinal cord or cauda equina. However, its absence does not lead to a more favorable prognosis when the neurologic examination indicates a severe lesion. The patency of the subarachnoid space is of importance for the course of subsequent treatment is often guided by it as emphasized by Coleman.[1] By compression of the jugular veins there is a prompt rise in cerebrospinal fluid pressure which is transmitted throughout the subarachnoid spaces. Obliteration of the subarachnoid spaces of the spinal canal prevents transmission of pressure changes below the level of the block when compression of the jugular veins is carried out. Thus, with a lumbar puncture needle in place and connected to a manometer, if no rise of cerebrospinal fluid pressure occurs with jugular compression, complete obstruction of the subarachnoid spaces exist (positive Queckenstedt test). Should there be a slow rise of the fluid level on compression of the veins followed by a slow fall on release, an incomplete block is said to exist. Should there be evidence of pressure on the cord or cauda equina, treatment in most cases should be directed toward its removal.

The Queckenstedt test is a valuable adjunct to the treatment of spinal injuries but like many tests, when accepted at face value, it can lead to hasty or inadequate treatment. A block may persist for several days or longer following reduction of a dislocation or even after laminectomy when, in most cases, it is known that the spinal cord or cauda equina is not seriously compressed. This probably results from torn membranes at the site of injury or from the subarachnoid space having been sealed off due to pre-existing pressure. It is seldom, therefore, that additional treatment is necessary unless paralyses increase or fail to subside as would be expected. Conversely, a negative Queckenstedt should not always be accepted as a contraindication for laminectomy. Not infrequently, fragments of bone from vertebral bodies or laminae exert pressure against the spinal cord or cauda equina without producing obstruction to the flow of cerebrospinal fluid. It is not our purpose to minimize the importance of the Queckenstedt test, but we do believe that it is not an infallible guide to treatment.

From the standpoint of treatment and prognosis, the cervical, thoracic and lumbar regions of the spine should be discussed separately as each presents individual problems.

Treatment of Cervical Injuries. Injuries of the cervical spine endanger life and function to a greater extent than those at other levels. Furthermore, due to the extreme mobility of the cervical spine, augmentation of spinal cord compression is not uncommon. Therefore, these injuries must receive immediate and adequate treatment.

Fortunately, the cervical spine lends itself admirably to traction. By means of gentle, skeletal traction most dislocations can be reduced within one or two hours, or sooner if heavy traction is applied. We have always preferred the former method because it is less injurious to the brachial plexuses and spinal ligaments. Although an hour or more may be required for complete reduction, decompression of the spinal cord begins almost immediately after the application of effective traction.

Perhaps there are a few circumstances, mostly theoretic, which would cause one

to discontinue traction and employ laminectomy. But during the fourteen years which have elapsed since we (W. G. C.) introduced skeletal traction,[2] we have not found any indication for laminectomy in the cervical region. In the past, massive extrusions of the nucleus pulposus, especially in the cervical region, probably have been overlooked. Perhaps some of the patients diagnosed as having had a recoiling dislocation were paralyzed as a result of a herniated nucleus pulposus. In our experience, all such cases without x-ray evidence of bone injury were completely paralyzed. Therefore, laminectomy if indicated would not have altered the prognosis. It seems certain, however, that the incompletely paralyzed patient should be given the benefit of exploratory laminectomy if he presents little or no x-ray evidence of bone injury together with a persistent block.

Uncomplicated anterior compression fractures of the cervical spine seldom require more than immobilization in plaster or a good adjustable brace for approximately three or four months. The more serious compression fractures, especially those with fragments jutting into the spinal canal should, in our opinion, be treated by means of skeletal traction for a period of at least four to six weeks before the application of a fixed support. In some cases, we have used traction for a shorter period but have kept the patients in bed for several weeks after the application of plaster. In our opinion, casts and other appliances are completely inadequate when strong support is required.

We have used small skull tongs for twelve years and have found them entirely satisfactory regardless of the pull required. The use of this instrument has been described by one of us (W. G. C.) in several previous publications.[3–6] Failures, usually due to the instrument slipping out, result from improper application, failure to tighten the clamp when the instrument is loose or from the use of defective tongs.* Other methods of applying skeletal traction have been described.[7–10] These consist of using either large tongs which prevent the patient from being turned on his side, or wires through the skull which, while effective, require some knowledge of neurologic surgery.

To obtain perfect or satisfactory alignment of the cervical spine is comparatively easy but to maintain it may be difficult. Dislocations of the atlas or axis almost invariably recur even after being held in a corrected position by means of traction for several weeks. Therefore, we believe that fusion, such as that described by Cone and Turner,[11] is indicated in these cases and that it should be performed without undue delay. Dislocations lower in the cervical spine are often associated with fractures of the pedicles which make it difficult to hold the vertebrae in alignment after the removal of traction. In recent years, we have continued traction in such cases from four to six weeks before applying plaster and allowing patients out of bed. This plan of treatment has been much more satisfactory than the earlier one which permitted early ambulation and we have seldom found it necessary to resort to spinal fusion. Many of the potentially ambulatory patients with dislocations below C-2 and with intact pedicles require only a short period of hospitalization. Usually, the deformity can be corrected and plaster applied within twenty-four hours. All of these patients, particularly those who are up and about, should be checked by x-ray examination at least once a week for a month or until it is determined that the corrected position will be maintained.

* During the war, it became necessary to have a large percentage of the Crutchfield tongs made by subcontractors, and, as a result, these instruments were defective. Instead of opening to a minimum of 10 cm. (between points with the instrument open), many had a spread of only 6.75 cm. which was inadequate except for very narrow skulls. This error has been corrected and the points have been improved as a further safeguard against excessive penetration of the skull.

Treatment of Thoracic Injuries. In the thoracic spine, compression fractures are the rule and the incidence of severe spinal cord injury is high, due to the comparatively small canal in this part of the spine. Because of its attachments to the rather rigid thoracic cage, the thoracic spine is seldom the site of dislocations. When they do occur, they are the result of extreme violence and almost invariably the spinal cord is hopelessly destroyed.

Simple compression fractures associated with little or no cord injury are mainly orthopedic problems. Hyperextension on a Bradford frame, followed by the application of a plaster jacket, usually is sufficient to bring about an entirely satisfactory result.

Fractures and dislocations associated with a complete transverse cord lesion require little in the way of specific treatment. Laminectomy is useless but it may be performed in selected cases to give the patient the benefit of a doubt especially when a spinal subarachnoid block persists. Also, laminectomy or removal of spinous processes may be indicated for the elimination of a bony prominence over which a pressure sore is apt to develop.

Laminectomy is the procedure of choice for decompression of the spinal canal in the thoracic and upper lumbar spine (through L-1). Laminectomy is indicated and it should be performed as soon as the patient's general condition will permit if he presents evidence of a severe but incomplete spinal cord lesion together with a subarachnoid block. When the spinal cord injury is slight and x-ray evidence of cord compression is lacking, immediate operation may not be altogether necessary even though a block exists. Unusual delay in neurologic recovery or certainly increasing paralysis would be an indication for laminectomy in such cases.

Treatment of Lumbar Injuries. The spinal cord extends to the lower part of the first lumbar vertebra; therefore, when discussing injuries of the lumbar spine, which generally offer a favorable prognosis, one should exclude the first lumbar vertebra for lesions at this level present the same problems as those in the thoracic spine. The lumbar canal contains the cauda equina which is composed of motor and sensory peripheral nerve roots. The motor roots have power of regeneration which accounts for the return of at least some neurologic function which occurs in the majority of patients with paralyses resulting from fractures and dislocations of the lumbar spine.

Simple compression fractures of one or more bodies of lumbar vertebrae, resulting from acute hyperflexion of the spine, are common and as a rule only hyperextension and the application of a cast are required. These injuries seldom disturb or endanger the cauda equina.

Severe compression fractures with fragments of bone displaced posteriorly often are associated with paralyses of the lower extremities, varying in degree. Usually, a spinal subarachnoid block is found in such cases. Decompression of the cauda equina by means of laminectomy followed immediately or later with spinal fusion especially if the patient is expected to recover motor function, is the procedure of choice in most of these cases. However, in certain cases showing only moderate posterior displacement of bone fragments, gradual hyperextension without laminectomy is justifiable. When this is done, constant attention should be given to the patient's neurologic status and the procedure must be immediately discontinued if increasing neurologic dysfunction is observed. If there is dislocation with or without fracture, this or any other part of the spine should not be hyperextended without first bringing about a reduction of the dislocation.

Occasionally, in the lumbar spine one encounters a complete dislocation with over-riding of the two ends. Needless to say, such injuries are incompatible with return of neurologic function regardless of the type of treatment employed. Many of

these patients live indefinitely and their neurologic deficit is no greater than in other patients with a good weight-bearing spine. Therefore, we have practiced open reduction in such cases to provide a weight-bearing spine and to remove bony prominences which endanger the overlying skin. Operation, of course, is always postponed until the patient's general physical condition is satisfactory.

GENERAL CARE OF THE PATIENT

The general care of the paralyzed patient should be well understood but so often it is woefully lacking in one or more details. This is understandable when one realizes how much constant nursing care and supervision these patients require especially during the first few weeks following injury. When pressure sores and urinary complications are prevented, primary hospitalization usually should not exceed two to four weeks. Then the patient can be transferred to his home, to a rehabilitation center or to a hospital equipped to take care of his prolonged or permanent needs. If complications especially pressure sores develop, the patient may not be moved for many months during which time both the patient and hospital sustain an enormous economic loss.

It is a common observation that private patients who can afford special nursing care almost never develop pressure sores, whereas ward patients almost invariably do. To protect patients and to utilize hospital beds to the fullest extent, our hospital authorities were made to realize the wisdom of providing special nursing care for all patients regardless of their financial status. During the several months that this plan has been in effect, no patient has developed a pressure sore and our paraplegics have remained on the service for a comparatively short period of time.

Loss of bladder and bowel control is one of the most serious and disturbing effects of spinal injuries, and unfortunately methods for restoring anything like normal or satisfactory control in the severely paralyzed patients have not been devised.

Management of the paralyzed urinary bladder varies in different clinics, but the objective is essentially the same, namely, to establish adequate drainage with minimum urinary sepsis until automatic function develops. The plan we use and find as satisfactory as any is as follows: An indwelling catheter is used in females. The same is used in males, for several days, until the prognosis as to life and function is determined. If the male patient has a complete or very severe cord lesion, suprapubic drainage is instituted. If the cord lesion is such that voluntary control may return within perhaps four to six weeks, tidal drainage such as that described by Munro[12] is employed. In both males and females mechanical drainage is discontinued when automatic bladder function appears, usually within four to twelve weeks. This is present when there is intermittent expulsion of urine through the urethra or around the tube. With proper timing and by exerting pressure over the lower abdomen, some of these patients get along reasonably well whereas others are less fortunate and must depend on using a receptacle strapped to the body or thigh. In all cases attacks of urinary sepsis are common and the attention of a urologist is indispensable.

Usually by regulating the diet together with the use of mild laxatives, such as mineral oil, the bowel contents can be kept at a semi-solid consistency and can be removed by enema. Unless this is done, either fecal impactions develop or the stools become so soft that soiling and maceration of the skin become very troublesome.

One of our most important problems has been the after-care of permanently paralyzed patients. With the establishment of paraplegic centers for veterans, it is now possible for many patients, primarily treated in civilian hospitals, to be transferred to these centers where specialized care, including vocational training, is

provided. Until recently the civilian, without a service record, had no place to go. Now, some states, of which ours is one, are opening rehabilitation centers which should offer some hope and encouragement for these unfortunate patients.

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Spondylolisthesis, though primarily an orthopedic problem, may progress to such a degree that changes in the nervous system require neurosurgical treatment. The most commonly reported involvement of the nervous sytem consists of compression of the cauda equina by the narrowed canal, resulting in varying degrees of sensory and motor loss in the sacral segments.

PREFRONTAL LOBOTOMY*

INDICATIONS AND RESULTS IN SCHIZOPHRENIA

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IT has been reliably estimated that approximately 40,000 new patients are admitted to mental hospitals each year with schizophrenia and that about a quarter of a million hospital beds in this country are constantly occupied by schizophrenics. Probably around no other mental illness has there been so much controversy and so much difference of opinion concerning its origin, etiology, symptom formation and treatment.[1] This has led one investigator to ask, "Is schizophrenia an adaptation or is it a disease?" How a patient with this condition can live on for years if not for decades and then come to necropsy with a brain that shows no indications of disease either grossly or microscopically is a challenging problem. Our approach to the problem has been neurophysiologic and our attitude has been pragmatic.[2]

Our first lobotomies for schizophrenia were performed in 1936 with immediate relief of symptoms, but within a few months several had relapsed. Some of these were reoperated upon but end results were modest. About the time our patients began to relapse, enthusiastic reports upon the effect of insulin shock therapy began to appear in the literature so we discontinued the operative treatment. Then gradually, as follow-up studies revealed that the early reports on shock therapy had been overly optimistic, we cautiously resumed the surgical treatment with an improved technic. When our book, Psychosurgery, was published in 1942[3] there were only twelve schizophrenics in the series; we now have approximately 250. It was about this time that Strecker, Palmer and Grant[4] showed that excellent results could be achieved in the chronically disturbed schizophrenics. This work was followed up on a large series of cases by Schrader[5] and others, so we followed their lead and achieved comparable success.

When a patient with schizophrenia is being considered for lobotomy, a number of questions must be answered: What symptoms are present which can be relieved? Is there any other method available to the individual which offers a reasonable hope of recovery? Is the condition serious enough to risk a major brain operation and all this implies? Will the family be satisfied with the modest result or do they anticipate a miracle? Can the family care for the patient during the convalescent period after he leaves the hospital?

Obviously not all schizophrenics will be improved by psychosurgery. The best results can be secured in those who show evidence of emotional tension. The emotional component can be judged by the severity of the complaints, the disturbances in behavior and the deviations in autonomic balance. When there is no history of outbursts of temper and assaultiveness, and the patient sits quietly in a corner during the interview listening with indifference to the voices, and examination reveals normal pupils, pulse and peripheral circulation, then the fight is over. It is too late for psychosurgery because a deteriorated precox behaves the same before and after prefrontal lobotomy.

THE PRECISION METHOD OF OPERATION

Prefrontal lobotomy is performed through burr holes made through the coronal suture and in the standard operation the path-

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ways are cut in the plane of the suture down to the sphenoidal ridge. In the more severe cases of longer duration the pathways are severed 6 to 12 mm. posterior to the sphenoidal ridge. Operation severs the thalamofrontal pathway and specimens show degenerative changes in the medial dorsal nucleus of the thalamus. Severing causes a reduction of disappearance of the emotional feeling tone that attaches itself to the ideas elaborated by the frontal lobes. The surgical problem is to place the incisions far enough posteriorly to reduce the charge to the point where the ideas no longer interfere with the patient's ability to work, yet not too far. Each millimeter of tissue behind this plane that must be sacrificed reduces the ability of the patient to adapt himself in a social environment.

Standard prefrontal lobotomy was performed in 63 per cent of the patients and the radical operation was used in 37 per cent. Twelve per cent required two or more operations to relieve the symptoms.

Nearly all of the patients who are referred for prefrontal lobotomy have had previous psychiatric care and shock therapy. Most have received electroshock and many have had insulin and metrazol. The number of shock treatments has ranged from 15 or 20 to over 200. The majority of patients have been hospitalized so it is obvious that they are disabled. A small minority come to us directly and these have received psychotherapy and electroshock. It is our opinion that most patients in whom shock therapy is effective begin to show definite improvement after six to ten treatments. If no improvement occurs after this and if there is an intense suicidal drive, institutionalization or lobotomy is recommended. Although a recent report from England covering 1,000 cases indicates that the results are much better when prefrontal lobotomy is done early in the course of the disease, we rarely employ it unless the symptoms have been present more than two years. If shock therapy gives a temporarily good result we are inclined to believe that lobotomy will give a prolonged good result. However, the fact that shock therapy fails does not mean that lobotomy will also fail. If an individual is not facing disability or suicide, a lobotomy is not indicated. The fact that a person exhibits a schizophrenic reaction does not constitute an indication for operation. As a matter of fact, when a whole family comes to the office for an interview, it is not always easy to pick the patient out of the group. However, we operate on the one who is disabled rather than the one with the craziest ideas.

In private practice it is almost as important to select the family as to select the patient, because they play such an important rôle in the convalescent care. They may have read an account of the operation or may have heard of some patient in the same institution who was operated upon with an excellent result. Naturally, they expect the same, not realizing that their son broke down under different circumstances and has now reached a stage of deterioration so that the most that can be hoped for is that he can live at home. They must be cautioned against expecting a full return to normal. It is our opinion that a patient who can go directly home ten days after operation has a much better chance of achieving a satisfactory social adjustment than one who must return to even a good mental institution. At home all kinds of pressure are brought to bear to make him behave normally. In an institution he is treated like a patient. If he wets or soils himself, someone cleans him up and then most likely he is transferred to a back ward where other patients are behaving in a similar manner and he will not be noticed. The lobotomized patient is like a child and the family is presented with an opportunity to train him over again. If he is allowed to develop bad or slovenly habits, the golden opportunity may be lost. It is true that the going may be difficult. Therefore, arrangements must be made prior to operation for some member of the family, preferably a mother, a sister or a brother who can devote his full time to the patient

for a period of at least three months and often six months. In taking the history, we inquire about the size of the house, the members of the family who live there, and what members of the family may be called on for assistance. The fact that some member of the family is willing to pay the hospital, special nurses and surgeon indicates considerable interest in the patient, so this is an immediate advantage.

As our studies in psychosurgery progress, the rôle of the operation itself will gradually fall into its proper perspective. In the beginning, it seemed probable that after cutting certain pathways in the frontal lobes, the patient would use what was left as best he could, and that would be that. It is almost that simple in older individuals with agitated or involutional depression of short duration. They go home a week or ten days after operation with the agitation and feeling of guilt gone, the emotional tension is reduced, they stop worrying about trifles, and if the family does not like the way the place is run, they can do it themselves.

In schizophrenia the problem is not so simple. At first we told the family that improvement might continue for six months after operation, then a year, and now we believe that improvement may continue for three or four years. It has become obvious that the majority of schizophrenics require psychiatric care or at least guidance following lobotomy. Some have been peculiar all of their lives and many developed symptoms at an early age and never had a regular occupation. They do not have a job to go back to like the obsessive compulsives, or housekeeping to resume like the involutionals. They have to develop new interests and these must be in line with their capabilities and opportunities. Instead of taking patients away from psychiatrists, it is more probable that psychosurgery will increase the number in need of psychiatric care as contrasted with custodial care. In mental hospitals lobotomy will reduce the number of special treatment rooms and camisoles, the euphemisms for padded cells and strait jackets, respectively. These patients who were violent, assaultive and altogether unapproachable before operation then become accessible for psychotherapy and occupational therapy.

At the present time, the patient can get more in the home than in most mental hospitals. The Veterans Hospital in Roanoke, Virginia, has started a program of rehabilitation for lobotomized individuals which may prove to be an important contribution. A psychiatrist, a clinical psychologist and a supervising nurse are assigned to the program. They study each patient and try to develop his special interests. One private mental hospital has already started construction of a five story pavilion for psychosurgery. It has facilities for training its patients which includes painting, appreciation of art, architecture, law and what not. With intelligent study of the patients it should prove an ideal place for re-education and rehabilitation. Naturally any such program must take into consideration the deficits produced by the operation, as we have a changed individual to deal with.

As stated earlier, our attitude is pragmatic. . . . We are more concerned with social adjustment and ability to work than with insight and ability to discuss personal problems. With the fantasy life smashed, dreams and hallucinations lose their importance and the individual can turn his attention to things going on around him. He is willing to undertake work commensurate with his ability and is more easily satisfied with his accomplishments. Only 2 per cent of the patients were keeping house before operation and none were employed. Of the 190 schizophrenics who have been followed a year or more after lobotomy, 17 per cent are regularly employed, 15 per cent are employed part time and 7 per cent are keeping house. This means that 39 per cent are usefully employed. An additional 33 per cent are living at home, more or less as drones because of inertia or insufficient relief of symptoms. Only 28 per cent are confined to institutions.

We now have enough information to discuss the prognosis with the family and if they expect no more than we can reasonably offer, then a prefrontal lobotomy may be indicated. Naturally if we think that the patient will be sufficiently relieved to get a job or resume housekeeping, we advise lobotomy. If he has been confined to a hospital for a number of years and the family would be satisfied to have him home if the assaultiveness could be relieved, then lobotomy is indicated. If a patient is so intensely suicidal or homicidal that he is confined most of the time to a strong room and unable to wear clothes or sleep on a bed, lobotomy is indicated even if the chances of leaving the institution are minimal. In fact, these are some of the most gratifying cases. To see an individual changed virtually from an animal in a cage to a person who lives with other patients on an open ward and eats with them in a dining room and walks out in the sunshine again even if he never leaves the institution, seems very worth while.

Since the decision as to whether or not a lobotomy should be performed ultimately depends on the prognosis, it may be well to summarize some of the more important factors which influence the prognosis.

The presence of *emotional tension* is the most important single factor. If it is present, we have something to offer; if it is absent, we have nothing to offer.

The duration of the symptoms and duration of disability are less important than we formerly thought. A follow-up study of 178 cases, followed for a period of one to ten years, reveals that good results may be obtained in 40 per cent and moderate improvement may be expected in an additional 30 per cent. The percentage showing improvement is about the same whether the disability has existed for two years or five years. This does not mean that the time factor is not important. We have in mind a young woman who appeared to be an excellent candidate for prefrontal lobotomy. By the time her family finally reached a decision two years later, she had deteriorated so much that operation hardly seemed worth while.

The younger the patient at the onset of symptoms the worse the results. An individual who breaks down in his thirties, expecially after some precipitating cause has a much better chance than one who breaks in his teens. The latter can frequently return to his job or resume housekeeping. Prognosis is worse in children.

It comes as somewhat of a surprise to find the hebephrenic schizophrenic responds just as favorably, if not more so, than the paranoid and catatonic. The probable explanation is that he is less aggressive and the convalescent care is easier to carry out; he is more likely to carry out suggestions and commands of his family or employer.

The operative mortality is 2 per cent. Twelve per cent of the patients develop convulsive attacks. If the patient's condition is not considered serious enough to warrant taking these risks, prefrontal lobotomy is not indicated.

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CONGENITAL ANOMALIES OF THE NEURAL AXIS*

SURGICAL MANAGEMENT BASED ON EMBRYOLOGIC CONSIDERATIONS

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PLANS for surgical intervention in a patient with a congenital malformation at any point along the neural axis should be directed toward improving the chances for normal future development and prevention of further deformity. When patients are born with visible malformations such as spina bifida or cranium bifidum, neurologic deficit may be apparent at once or may develop only after a period of growth. Patients in both categories may have their condition ameliorated or cured by judicious surgical intervention or orthopedic care. It follows that the ability to make an estimate of the embryologic deficit of a patient is as important as gauging the degree of the neurologic deficit. Growth not only may augment the deformity but may also render irreversible changes which were once reversible. Therefore, judgment in the selection of individuals for surgery, as well as the time and type of operation, should be based upon embryologic as well as neurologic knowledge. One should not attempt to correct an insurmountable anomaly only to leave a child unable to become physically and economically independent.

Existing knowledge of human embryology is almost wholly limited to morphologic description of the normal and the abnormal. Because great technical difficulties hamper experimentation in the mammalian field, the most extensive experimental advances have come from work on amphibians and the chick. Conclusions based on investigation in one species can be applied to different species only with reservations. Nevertheless, the recapitulation of phylogeny by ontogeny permits conjecture that some of the basic mechanisms of embryology in lower forms must carry over, at least in principle, in man. Consequently, a brief discussion of the factors controlling embryonic development is in order.

The blastopore of an early embryo is a collection of totipotential cells. The three primitive germ layers, ectoderm, mesoderm and endoderm which arise from this center of growth extend cephalad in contrast to the primitive streak which lies caudad. Formative movements, such as invagination, lateral expansion, longitudinal extension and dorsal convergence, so shape the interstitial growth of tissues derived from the primitive germ layers, that further morphogenesis leads to completion of the embryonic system. Spemann[1] questions whether the faculty of formative movement in embryonic tissue may not be determined as early as fertilization in vertebrates. He later suggests that harmony of decisive extrinsic factors is necessary to stimulate integrated movements which are intrinsic qualities of competent tissue. In embryologic parlance, this process is termed induction. Needham[2] considers tissue to be competent when it is able to react to a given morphogenetic stimulus in such manner that determination of histogenesis will lead to proper organogenesis. Workers in Spemann's laboratory[3] have achieved induction of neural tissue from undetermined ectoderm of amphibia by the application of a chemical extract of tissue excised from the region of the blastopore of other embryos. This leads Spemann to conclude "that inductive action is trans-

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ferred by a chemical substance." In the embryologic literature such chemical substances are referred to as *organizer substances*. An integrated and synchronized system of induction appears to reign during the elaboration of the embryo, with each system and its integral parts adhering to a strict schedule. From experimental evidence, Spemann[4] points out that tissues from which the neural structures arise are not susceptible of induction until after the mesodermal substratum has formed, and then for only a brief period. Gilchrist[5] by an ingenious method of directing heat to any given point on the neural axis of incubating newt's eggs, demonstrates that there are temporal limits for determination of individual elements of the neural axis. He further determined that a thermal disturbance applied within such temporal limits so alters the physiology of the part affected that the local pattern of determination will be abnormal.

If the embryo is inspected at the stage of invagination of the blastopore (gastrulation), differentiation of the three primary germ layers is apparent as the preface to future organogensis. Interruption at the next stage, neurulation, reveals elaboration of the neural axis. At this time, the general pattern of the embryo may be foreseen in the concomitant somite formation. A slow-motion viewing of this process reveals that the ectoderm from which the central nervous system is derived appears first as a groove between two rising crests. Needham[6] points out from avian studies that the blastopore moves backward, followed by the folds of the neural crests, "as if it were drawing the latter as a pencil would do." Bremer,[7] on the other hand, believes that the neural axis grows primarily forward from the neuropore, thus causing the projection of the head. The folds converge dorsally to form the neural tube by fusion. In man, fusion of the neural tube is initiated between the twenty-first and twenty-second day after ovulation. Bremer[8] considers this initial point of fusion to be the site of the caudal end of the hindbrain. Normally, fusion is completed at the cephalic pole of the neural axis between the twenty-fifth and twenty-sixth day and caudally by the twenty-ninth day. As closure of the neural tube progresses, concomitant encasement with mesenchyme causes it to appear to sink beneath the surface ectoderm. Mesenchyme derived from the mesoderm of the supporting somites, in addition to supporting and insulating the neural tube, has the prime function of inducing it. It is at this crucial embryonic stage,[8] the twenty-first to twenty-ninth day after ovulation, that the groundwork is laid down for malformations, such as spina bifida and cranium bifidum with or without meningeal protrusion. Myelomeningoceles and encephaloceles accompanying spina bifida and cranium bifidum may be construed as arising from faulty induction of ectoderm, secondary to the mesenchymal defects. Patten[9] reports two human embryos, aged seven and six weeks, respectively, with malformations suggestive of encephalocele and myelomeningocele formation. He believes that lateral overgrowth of neural tissue preventing dorsal convergence is the cause of failure of closure of the neural tube in these cases.

For purposes of discussion, it is preferable to look upon the conditions found in Patten's two embryos, and all neural malformations along the neural axis, as abnormalities of neurulation. Bifid conditions, cranial or spinal, with or without meningeal protrusion in conjunction with abnormal overgrowth of superficial blood vessels or fat may be considered the result of faulty mesenchymal induction. Gastrulation and neurulation depend upon the essential morphogenetic movements termed invagination and dorsal convergence, respectively. Exogastrulation in amphibia, through reversal of invagination, has been produced at will by Holtfreter,[10] and Curtis et al.[11] The former achieved this reversal of formative movement by maintenance of amphibian blastulae in 0.35 per cent saline solution, while the latter achieved similar results by irradiation. These environmental

abnormalities apparently interfered with the reaction of organizer substances and competent tissue in such a manner as to reverse the direction of morphogenetic movement.

Recently, Ancel[12] has observed myelomeningocele formation following inoculation of fertilized chick eggs with various chemical agents between the sixteenth and twenty-sixth hours of incubation. He has achieved the same result by incubating the eggs at 40.5°C. instead of 37°C. His experiments reveal that the earlier chemical or physical disturbances take place, the higher the deformity will lie on the neural axis. He concludes that myelomeningocele formation is dependent upon faulty mesenchymal induction.

Snell, Bodeman and Hollander[13] have produced gross abnormalities of the central nervous system, similar to that described by Patten[9] as the precursor of an encephalocele by irradiation of male mice prior to mating with normal females. In this instance, it might be construed that the genetic change which followed irradiation had rendered the tissue incompetent to respond in a normal manner to the morphogenetic stimuli or the organizer substances incapable of evoking appropriate stimuli.

In man, the cause of spina bifida, cranium bifidum, meningeal and neural malformations is not known. The experimental work on amphibia, birds and lower mammals mentioned above suggests that possibly, in man, similar aberrant factors may adversely influence neurulation. Thus, abnormalities of neurulation may result from incompetence of mesoderm and ectoderm, or the reaction of organizer substances so altered in quality or time or action, that the resulting stimulus leads to abnormal induction. The extent of the anomaly will depend upon the degree and duration of imbalance and the area in which it operates. Therefore, the protean forms of spina bifida, cranium bifidum with vascular, fatty and meningeal defects as they are seen clinically, may be quite reasonably postulated in view of the multipotential nature of mesenchyme. It should be remembered that mesenchyme is a primitive tissue from which corium, fat, fascia, muscle, blood vessels, the components of the vertebral column, notochord, chondral and membranous portions of the skull and some portions of the meninges are differentiated but not the neural elements.

It is now appropriate to examine the embryologic evidence which will throw light upon the anomalies, ranging from the minor myelomeningocele and encephalocele, through anencephaly. In these anomalies there is neuroectodermal malformation in addition to the mesenchymal defect.

Exogastrulation studies such as Holtfreter's[10] have demonstrated that with complete evagination of the blastopore (exogastrulation) no neural tube differentiation is possible. Needham,[14] in discussing exogastrulation, makes the statement that, "The underlying of the dorsal ectoderm by mesoderm is the full, perfect, and sufficient cause for its determination for neural differentiation."

Holtfreter's[15] later work with explanation of not fully determined tissues further confirms Needham's hypothesis. Holtfreter discovered, as is illustrated in Figures 1 through 5 that proper neural induction is dependent upon normal development of mesoderm.

Conditions depicted in Figures 2 and 4 are suggestive of hydromyelia in man; likewise Figure 3 is suggestive of myleomeningocele formation as seen in man. Possibly the factors producing these abnormal inductions under experimental conditions may exist in the form of mesodermal deficiencies in the human embryo when faulty neural induction leads to hydromyelia and myelomeningocele. In other words, clinical lesions ranging from a minor neural malformation associated with bifidism of the neural axis to complete rachischisis of the spinal column with dysraphism, or anencephaly, will depend upon the degree in which the mesenchyma is defective and, therefore incapable of proper neural induction.

A detailed discussion of the origin of teratoma and teratoid tumors is beyond the scope of this paper because of their neoplastic nature. However, because of their frequent association with congenital anomalies of the neural axis, they deserve mention here. The differences in life history and histological nature of the teratomas arising in the sacrococcygeal region as compared with those found elsewhere along the neural axis, makes it feasible to consider them separately.

The sacrococcygeal group of tumors are truly teratomatous in nature, constant of position and massive in growth with a high state of cellular differentiation at the time of birth. These characteristics suggest that they may result from tissue incompetence, or disturbance of organizer substances occurring at the blastopore when induction of the neural axis is being completed.

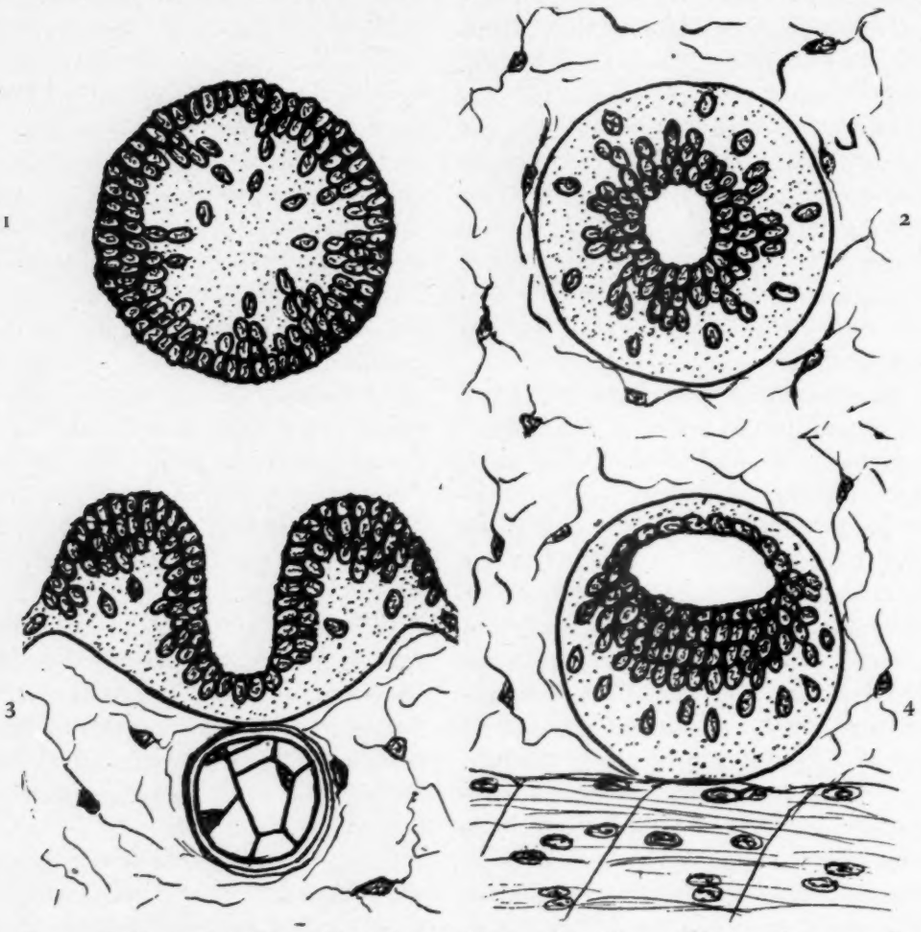


Fig. 1. Explantation of neural ectoderm without mesenchyme yields a sphere or rod with nuclei at the exterior.
Fig. 2. Induction of neural ectoderm within mesenchyme without notochord yields a hollow sphere or rod with nuclei centrally located.
Fig. 3. Induction in the presence of notochord with insufficient mesenchyme yields failure of closure of the neural tube with cell bodies lying along the free surface and thinning of the neural plate in contact with the notochord.
Fig. 4. Induction on muscle yields a tube with eccentric lumen.

Holtfreter's[10] work with amphibia suggests that exogastrulation, causing dislocation of essential toti- and multipotential cells, plays an important part in teratoma and teratoid formations along the neural axis. Exogastrulated amphibia developed teratoid growths in his experiments at the time that neurulation should have been taking place. Ingraham and O. T. Bailey's[16]

study of teratoma and teratoid tumors in man reveal evidence of congenital anomaly in several cases. This might be attributed to faulty neurulation following a disturbance in gastrulation. Further speculation may be considered futile in view of ignorance of the cause of neoplastic formation.

Persistence of remnants of the neurenteric canal is an extremely rare anomaly. Bremer,[17] in a review of the literature, found but three cases and attributes them to persistence of accessory neurenteric canals. Since the cases reported in the literature and the ones studied in this clinic have all been associated with spina bifida, it seems appropriate to introduce them in this discussion and to consider the possibility that they may result from an aberrant morphogenetic movement occurring late in gastrulation or early in neurulation.

Avian and human individuals both show incidental occurrences of spina bifida. Ancel's[12] studies reveal a 0.25 per cent incidence of this condition in normal control chick embryos. Ingraham and Lowrey[18] report from this clinic that approximately 25 per cent of normal children have bifid spinal conditions which are incidental findings in x-ray films taken for some other reason. Whether these variations from the normal are the result of adversive genetic action or alterations in the environment of the hen's egg or the human uterus, is a question that is far from settled. Murphy,[19] after extensive statistical and environmental study of families possessing malformed children, comes to the general conclusion that, "Gross human congenital malformations arise solely from influences which affect the germ cells prior to fertilization." In the course of his research, he was able to demonstrate that a congenital malformation will occur with twenty-five times greater frequency than that of the general population when an offspring is born to parents already possessing a malformed child. The more recent report of Gregg[20] tends to offset the pessimism of Murphy somewhat. The latter author shows an association between development of rubella in the mother and the occurrence of congenital ophthalmologic and cardiac defects in the child. This leads him to the speculation as to whether the cells most actively engaged in crucial points of organogenesis may not be so affected by the disease proc-

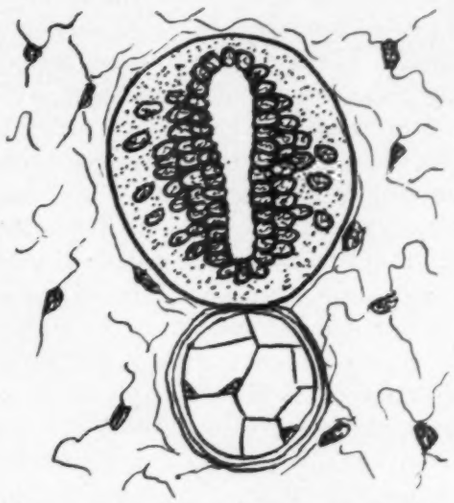


Fig. 5. Induction in the presence of sufficient mesenchyme and in contact with notochord yields a neural tube of proper contour.

ess that aberrant development results. Ingraham and Swan[21] report from this clinic in a study of 277 patients with spina bifida, that the family histories revealed a 6 per cent incidence of spina bifida and approximately the same incidence for other congenital anomalies. It is interesting to note that more recently in this clinic a few cases with major neural axial deformity have been recorded with a negative family history for congenital deformity, whose mothers and attending physicians can substantiate the fact that an acute upper respiratory infection with associated hyperthermia to 104–105°F. developed during the third and fourth week of gestation. In light of present knowledge it is doubtful whether the human intrauterine environment can be influenced by factors other than temperature without having the embryo so altered that it becomes spontaneously aborted. The research of both Gilchrist[5] and Ancel,[12] on lower invertebrates, which shows that the morphogenesis

of the neural axis may be affected by hyperthermia presents an interesting correlation with Gregg's[20] clinical observations. Needless to say, many more cases of proven hyperthermia at the time of neurulation, similar to the few recently studied in this clinic, will have to be accumulated before the pessimism of Murphy's conclusion can be lifted. However, it is to be hoped that other members of the profession will take an interest in this subject in order that a true evaluation of the causative factors of congenital anomalies, genetic and environmental, may be obtained. Undoubtedly, certain matings are genetically doomed to produce congenital anomalies of the neural axis, but it is possible that the occurrence of a certain percentage is due to a fortuitous circumstance such a hyperthermia coincident with neurulation. When this tremendous problem has been solved, doctors may, with more assurance, advise parents concerning the desirability of having additional children after the birth of a deformed offspring.

SURGICAL CONSIDERATIONS

Bearing in mind the foregoing comment on basic embryologic factors that may form the background for the origin of congenital malformations, attention will be turned to the surgical aspects of this discussion. Tempering of neurologic judgment with an embryologic concept will aid the surgeon in the selection of cases for operation as well as the time for and type of operation. Embryologic data pertinent to the anomaly under consideration will be presented wherever feasible. By careful individual evaluation, Ingraham and Swan[21] show in a series of 546 cases with spina bifida and cranium bifidum that 30 per cent of the former and 34 per cent of the latter may expect a relatively normal existence when aided by appropriate surgical measures. These figures tend to allay the pessimism of the past concerning the prognosis of patients born with neural axial deformities.

GENERAL ANATOMIC CONSIDERATIONS OF SPINA BIFIDA

The supporting structures of the neural axis are differentiated from mesenchyme which is, itself, mesodermal in origin. If the mesenchyme which develops parallel to the neural crests fails to encase completely the neural tube by dorsal or ventral fusion, spina bifida occurs. Dorsal spina bifida is much more common than the ventral type. Meningeal protrusion through the bifid defect constitutes a meningocele. This phenomenon probably occurs when all encasing mesenchymal elements, except the meninges, fail to fuse. Myelomeningoceles in man arise in association with spina bifida. Ancel[12] concluded from his experimental work that myelomeningocele occurs in the chick in conjunction with spina bifida as the result of faulty induction of neuroectoderm by mesoderm which in itself is so inadequate as to lead to spina bifida. That this may possibly be the case in man is not hard to conjecture.

The results of Holtfreter's[15] explantation experiments, in which he encased medullary plate of amphibians with mesenchyme, point out that neural induction in amphibia depends upon a mesodermal factor. Certainly the configuration of some of his abnormal inductions achieved by varying the amounts and components of mesenchyme, are suggestive of hydromyelia (Fig. 6) and myelomeningocele formation seen in man. Diplomyelia (Fig. 7), which occurs occasionally in patients with myelomeningocele, is thought to be a manifestation of partial twinning by Herren and Edwards.[22] Bremer,[8] in discussing gastrulation in amphioxus, the lowest form of chordate, points out that the nervous tissue lying in either lip of the gastrula mouth, later known as the neurenteric canal, becomes the homologue of the paired neural plates from which the central nervous system is developed. He later states that in higher vertebrates such as amphibia, gastrulation is passed through so rapidly that the paired neural plates on either side

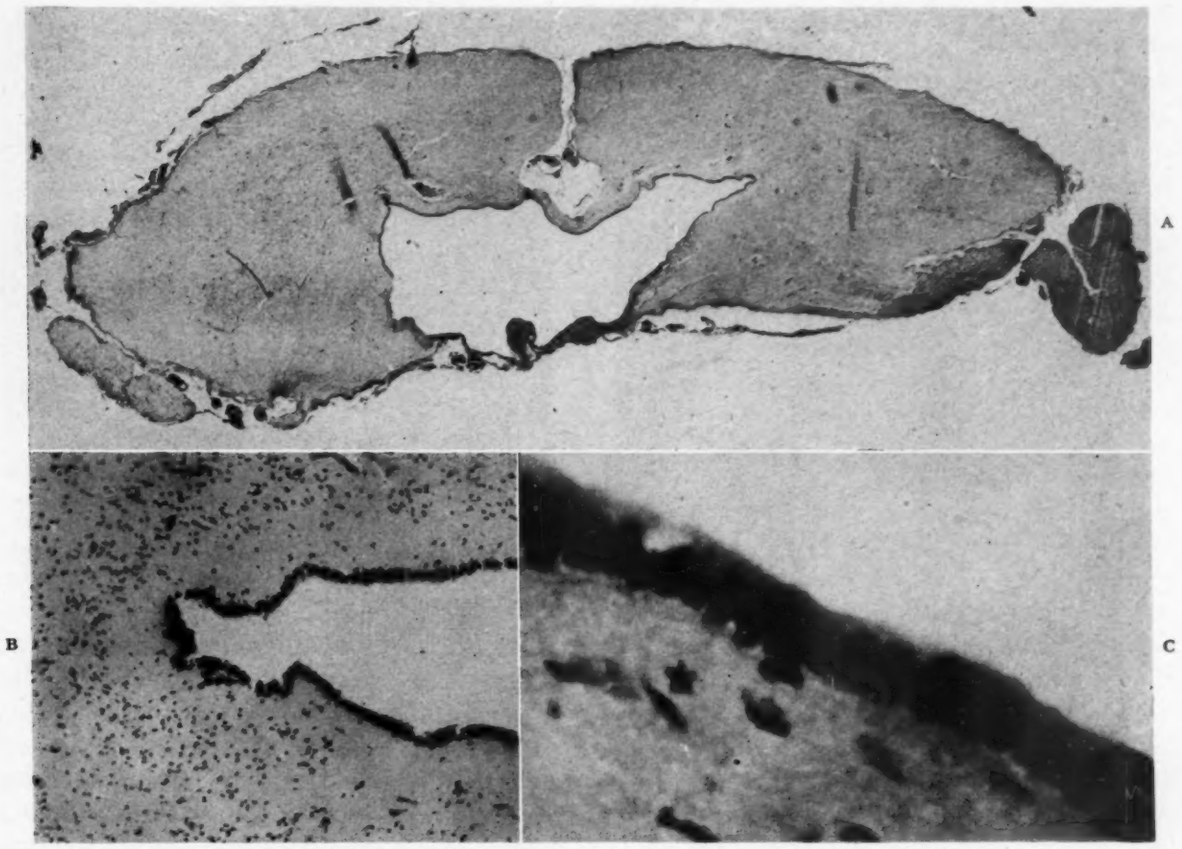


FIG. 6. A, photomicrograph of a high thoracic section of spinal cord showing hydromyelia. Hematoxylin and eosin stain. Magnification 15 diameters. B, magnification 130 diameters to show ependymal lining of the cavity. C, magnification 500 diameters to show further detail. This patient had a successful resection and repair of a thoracic myelomeningocele at the age of eleven months. Death resulted from an intercurrent gastrointestinal infection five weeks after operation.

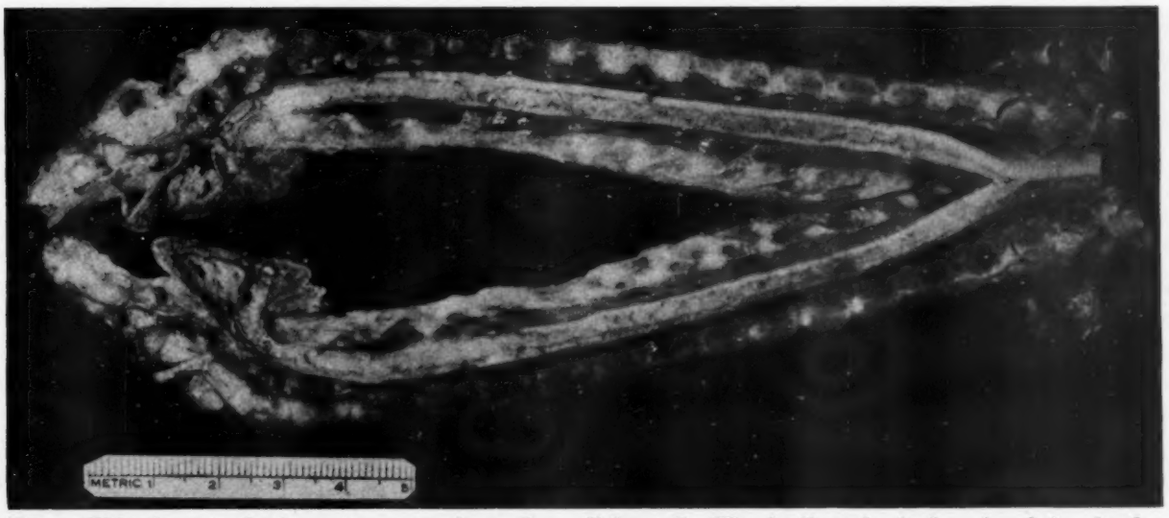


FIG. 7. This photograph of an autopsy specimen shows diplomyelia. The duplicated spinal cord ends in a lumbosacral myelomeningocele.

FIG. 8. This photograph reveals hypertrichosis over the site of a spina bifida occulta in a girl aged five years.

of the neurenteric canal giving rise to the nervous system, appear only as a fused neural groove growing forward from the blastopore. This author mentions experimental work in which the ovum is so damaged that the gastrula lips pursing the neurenteric canal do not fuse. Such situation, he points out, leads to diplomyelia in lower forms. With this experimental evidence at hand, it may be possible to imagine that diplomyelia in man may arise as a result of some morphogenetic disturbance in the stage of gastrulation.

Mesenchyme contains the mosaic for the fat, blood vessels, fascia, ligaments, bones, corium and some elements of the meninges. It is, therefore, not surprising that when spina bifida occurs, both fatty and vascular growths as well as aberrant fibrous strands should develop concomitantly with the bony defect. Abnormal fat overgrowths developing simultaneously with meningoceles and myelomeningoceles are so common that Ingraham and Swan[21] refer to

FIG. 9. This photograph reveals a dermal sinus situated in the upper sacral region over a spina bifida occulta. The dermal sinus which led down to an intrathecal teratoid tumor was surrounded by telangiectases.

these conditions as lipomeningocele and lipomyelomeningocele. Spina bifida occulta is a common defect in the general public. Ingraham and Lowrey[18] report a 25 per cent incidence in normal children. This condition obtains clinical significance only when neural elements are impinged upon by aberrant bone formation, fatty and vascular masses, or when constricted by adhesions. Surface manifestations of spina bifida occulta include hypertrichosis (Fig. 8), lipoma and angioma formation, dimpling of the skin, dermal sinuses (Fig. 9) or bony protrusions.

SURGICAL INTERVENTION IN SPINA BIFIDA OCCULTA

Bremer,[23] quoting his teacher, C. S. Minot, states that from a relative point of view, 90 per cent of growth and development is accomplished *in utero* when one considers what transpires between zygote formation and term as compared with term and full maturity. It follows then that surgery has little to offer the patient born with a major deformity and marked neurologic deficit, prenatal in origin. Such lesions may be looked upon as "fixed." On the other hand, patients born with a minor deformity who show increasing neurologic deficit as a result of interference with the dynamics of

ascent of the spinal cord after birth may be considered to be in a plastic state. The possibility of amelioration by surgery is far greater in this group provided it is carried on while the physiologic changes are still reversible.

At this juncture an account of the mechanism of ascent of the cord will be useful in illustrating why some neurogenic deformities occur and in selecting individuals for treatment. The caudad formation of the neural axis during the second month of gestation progresses so far that the blastopore finds itself at the tip of a free tail. This structure likened to a tail is a coccygeal continuation of the spine, complete with hind gut, containing spinal cord supported by somites and notochord. The primitive streak, in the meantime, has sought the ventral surface of the embryo from the tip of the tail to the body stalk. Toward the end of the second month, fusion of the vertebral elements of the tail occurs with concomitant ventral retraction within the soft tissues to form the adult coccyx. At this time, by virtue of vesiculation and degeneration, all coccygeal neural elements up to the first coccygeal nerve so deteriorate as to be recognizable only as the filum terminale. The hindgut likewise becomes fragmented and is absorbed. From the third month on, the spinal column grows more rapidly than the cord. This discrepancy in rate of growth normally leads to ascent of the cord through traction exerted by the relative fixation of the structures of the hindbrain within the calvarium. Under ordinary circumstances, at term, the tip of the conus medullaris arrives at the third lumbar level through constant traction. Therefore, the degree of faulty mesenchymal induction will determine the range and development between an inconsequential bifid defect and a massive one with aplasia and dysplasia of many of the supporting elements of the vertebral column. Varying degrees of neurologic deficit will occur in direct proportion to the distortion of the cord by adhesions, osseous and fatty masses. Acceleration of the rate of growth will increase the deficit because of the unbalanced traction exerted by ascent of the cord. It follows then that a severe degree of neurologic deficit may exist at term. Thus, neurologic deficit may never exist, or may vary from a minor sensory, motor or trophic change to a para- or quadriplegia with sphincter incontinence. Some of the commonest signs are underdevelopment of a foot or extremity, maldevelopment such as a valgus, varus or cavus deformity of a foot or weakness in a major muscle group of a lower extremity. The commonest symptoms are disturbances of gait, station or posture. Trophic disturbances with signs of sensory defect are seen as well. Failure to respond to bowel training or loss of previously established bowel and urinary control points to involvement of sphincters.

In infancy and childhood many valuable signs can be detected by observation of the patient at play or in moments of struggle. In addition, a detailed orthopedic and neurologic examination is possible with but few limitations because of age. Reflex changes can be elicited with reliability. As far as sensory deficit is concerned, failure of withdrawal and lack of vocal or lacrimal response to painful stimulation is reliable in the very young. Distraction with a nursing bottle or toy is useful as an adjunct to this examination in order that the patient may become quiet between stimuli. It is the experience of this clinic that no patient is too young for evaluation.

X-ray examination of the spine and extremities is useful in determining what particular bony abnormality is present. (Fig. 10.) The limitation of interpretation of spinal x-ray studies, however, is in direct proportion to the amount of calcium laid down in the chondral scaffold of the age group in question. Air and pantopaque myelography is a useful adjunct when the presence of an intraspinal defect or mass is in question. (Fig. 10.) Air is used in the younger age group to avoid the necessity of withdrawal of the contrast medium. In older infants, 1 to 3 cc. of pantopaque is recommended. A general anesthetic is

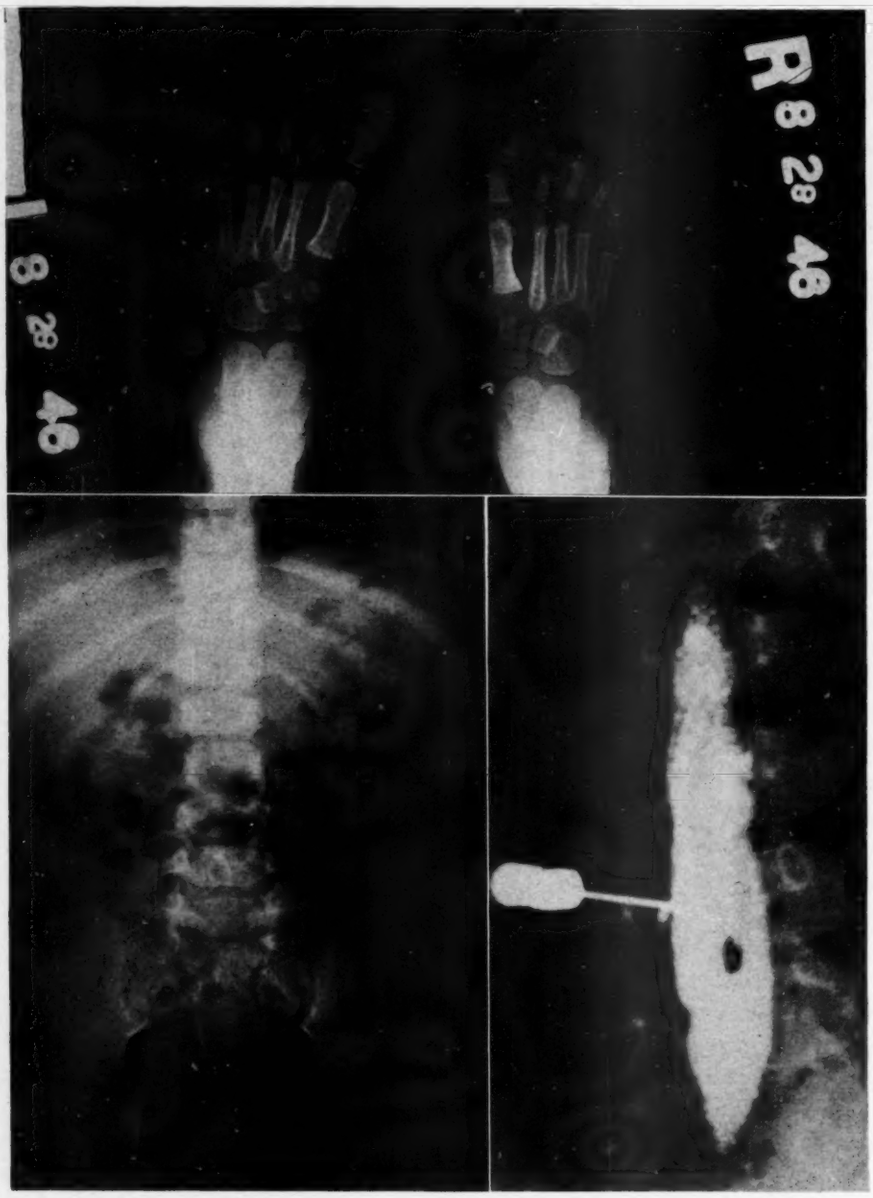


FIG. 10. A, photograph of roentgenogram of the feet of a patient aged one and one-half years born with a lumbar, sacral and coccygeal spina bifida occulta. Note the underdevelopment of the right foot as compared with the left. B, photograph of roentgenogram revealing the spina bifida occulta. C, pantopaque myelogram revealing abnormal bony spinule situated in the midline at L3. At operation this was found to be adherent to and impinging upon the right half of the cauda equina.

necessary to permit safe handling of the column of this contrast medium and to avoid spillage into the basal cistern and intracranial subarachnoid spaces. Introduction and withdrawal of the contrast medium is handled in the manner described by Kubik and Hampton[24] Urethrography, cystography and cystometry are of aid in distinguishing genitourinary tract anomalies from bladder disturbances of neurogenic nature.

PREOPERATIVE PREPARATION

In general, preoperative preparation of patients with spina bifida and cranium bifidum follows the principles outlined below. Fluids are restricted for at least six hours before operation; enemas are given

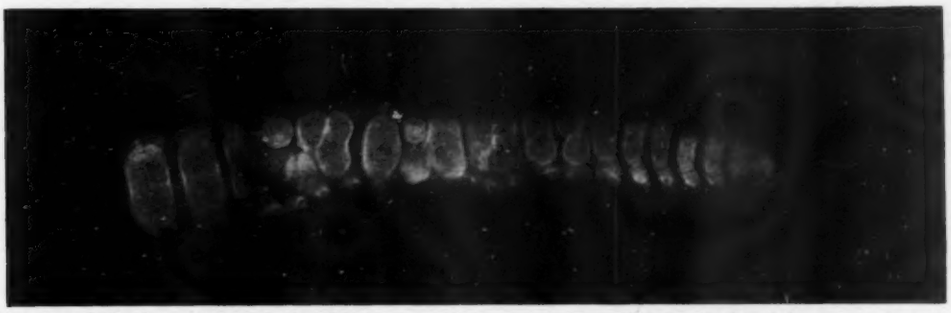


Fig. 11. This is a photograph of an x-ray of an autopsy specimen obtained from a five and one-half weeks old infant born with a myelomeningocele and spina bifida. Note the multiple bony anomalies in portion of the spine shown.

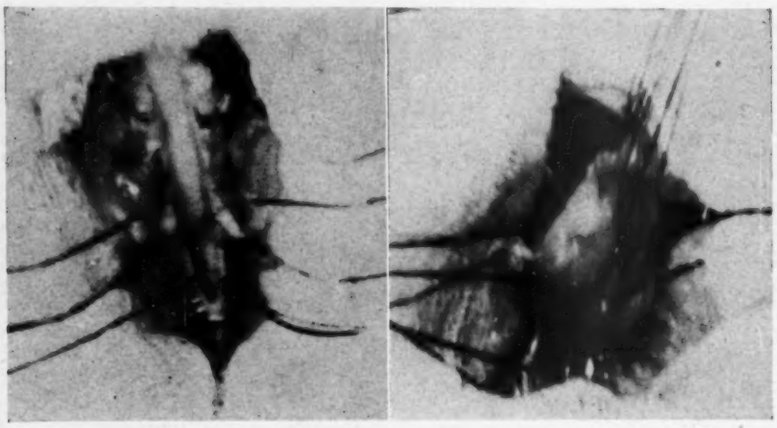
the night preceding operation in order to facilitate the absorption and administration of avertin. Sulfadiazine and penicillin are administered prophylactically when the operative site lies in the lumbar region or below, or when the crevassing or the granulations on the surface of the meningocele will not permit confidence of sterilization of the operative field with the usual chemical agents. Great care must be given to walling off the rectum from the operative field when a low exploration is planned. This is carried out before the skin preparation starts. No major procedure is started without the insertion of a constant intravenous cannula.

ANESTHESIA

In this clinic, most operations are carried out under avertin-ether anesthesia, supplemented by local infiltration of 0.5 per cent procaine with adrenalin 1:1000 added to the amount of 0.3 cc. to each 30 cc. Avertin is administered to children in the dose of 100 mg. per Kg. of body weight, and 80 mg. per Kg. of body weight in infants. Avertin is not administered to the newborn or to patients suffering from cachexia, renal or hepatic disease or to patients upon whom a low spinal exploration is contemplated for fear of contaminating the incision during operation. Intratracheal ether is administered with cervical or intracranial lesions in which embarrassment of the respiratory center during operation is likely. Anesthesia in the newborn is given in the form of open drop ether or brandy 4 to 5 cc. by stomach tube in addition to local procaine infiltration. When the infant awakens during the procedure, a nipple stuffed with cotton saturated with brandy diluted 1:8 is offered. This usually leads to further relaxation through raising the blood alcohol level.

SURGICAL TREATMENT OF SPINA BIFIDA OCCULTA

A midline incision is made from the first spinous process above and below the bifid area, after having been so draped that extension cephalad and caudad is facilitated if indicated. The laminae of the first normal superior and inferior vertebrae should be exposed before carrying the incision beyond the superficial fascia in the anomalous area. Great care must be exercised to prevent entering the dura or possibly the cord during dissection. Every adhesive band, until proved otherwise, should be looked upon as a peripheral nerve root. Findings at operation vary tremendously. Anomalous osseous arrangements involving the spinous processes, laminae, pedicles, facets and vertebral bodies are encountered in any combination. (Fig. 11.) Five cases of aberrant midline spicules of bone attached to the dorsal surface of a vertebral body impinging upon nerve roots, are on record in this clinic. Reversal of clinical signs in "nonfixed" lesions can be obtained by careful and judicious removal of these bony malformations with due regard being given to avoiding root evulsion and laceration of the cord. Fatty malformation, subcutaneous, extradural and intradural, is frequently encounted in the surgery of spina



A B

FIG. 12. A and B, these photographs were taken when the patient was two years old during the operative removal of an intrathecal lipoma shown in C.

FIG. 12. C, The lipoma was lumbosacral in position. The presenting symptom was poor rectal sphincter control. There were no neurologic signs except a questionably weak rectal sphincter. An upper sacral subcutaneous lipoma overlay a spina bifida occulta. 3×.

bifida occulta. A discrete tumor can be removed with reversal of signs caused by its impingement, provided intervention is timely. (Fig. 12.) Unfortunately, all lipomatous formations are not discrete. However, decompression afforded by exploratory laminectomy will relieve pressure and alleviate signs resulting from the extradural type. Likewise, decompression benefits the patient with diffuse lipomatous infiltration of the cauda equina and cord itself. Fibrous tracts, possibly sclerosed meningoceles or dermal sinuses lead down through the dura to the cord. Lysis of adhesions associated with the latter type of malformation, as well as with the lipomatous and osseous anomalies, may afford the greatest benefit because of the relief from the distortion of unbalanced traction placed on the growing spinal cord and nerve roots.

SURGERY OF MENINGOCELE AND MYELOMENINGOCELE

The clinical distinction between a meningocele and a myelomeningocele is most difficult and is more accurately made during operation or when pathologic studies are complete. Ingraham and Swan[21] adopted the following clinical criteria: "If a sac exists clinically and nerve elements cannot be seen, and if there is no evidence of neurologic disability as demonstrated by muscular weakness or paresis, loss of sphincter tone, or aberration in cutaneous sensation, the lesion is a meningocele. (Fig.

13.) If nerve elements are visible or neurologic disability exists, the lesion is a myelomeningocele. (Fig. 14.)" As has been shown before, meningocele and myelomeningocele formation is contingent upon a bifid condition of the spine. The former probably arises as the result of improper induction of all mesenchymal elements down to those containing the mosaic for the meninges; the latter may be attributed to abnormalities of neurulation involving the ectoderm as well. Abnormalities of neurulation of greater severity may account for peripheral nerve aplasia due to neural crest malinduction, hydromyelia or diplomyelia.

Therefore, in selection of patients for surgery and determination of the appropriate time for operation, the prevention of meningitis must be considered as well as the criteria used in cases of occult spina bifida. At the same time, the over-all prognosis is poorer when a myelomeningocele exists. This implies the addition of ectodermal malformation to the mesenchymal one of the accompanying spina bifida. Position, size, contour, condition of the herniated meninges and availability of tissue for closure are all factors that must be evaluated before attempting operation upon a meningocele. Thus, thin, pedunculated meningoceles with small stalks should be submitted to surgery as early in the neonatal period as the condition of the patient will permit, provided the patient does not in another way appear to be defective. Rupture of the sac, during delivery or soon afterward, is an indication for immediate intervention, as is incipient ulceration. In such cases, prompt surgical attention, combined with chemotherapy, has yielded gratifying results. Unfortunately, the broad based sessile type of meningocele is more common than the pedunculated. Inadequate tissue for closure of this type of defect, or frank ulceration precludes surgery in spite of the risk of meningitis from rupture or further ulceration of the membranous covering. Under these circumstances, protective dressings should be ordered. These consist of a layer of sterile perforated cilkloid over the surface of the meningocele and an outer layer of gauze. Around this a protective ring of sheet wadding, wrapped in gauze, is maintained in position by a binder. Epidermis propagates beneath the perforated cilkloid to yield the necessary protective barrier. Daily, and more often if indicated, the dressing is taken down to the cilkloid

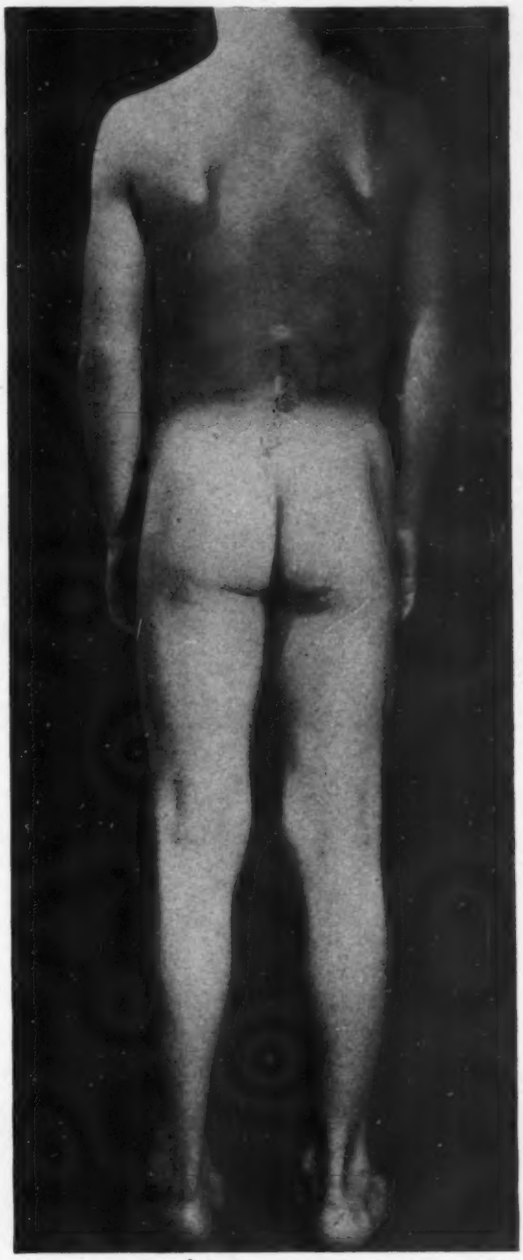
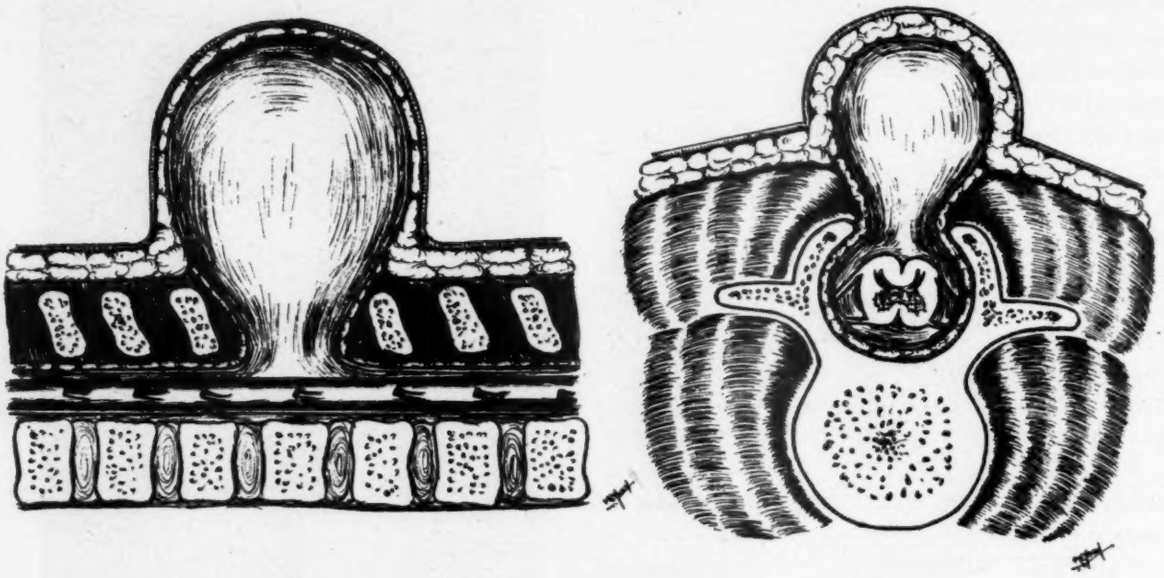


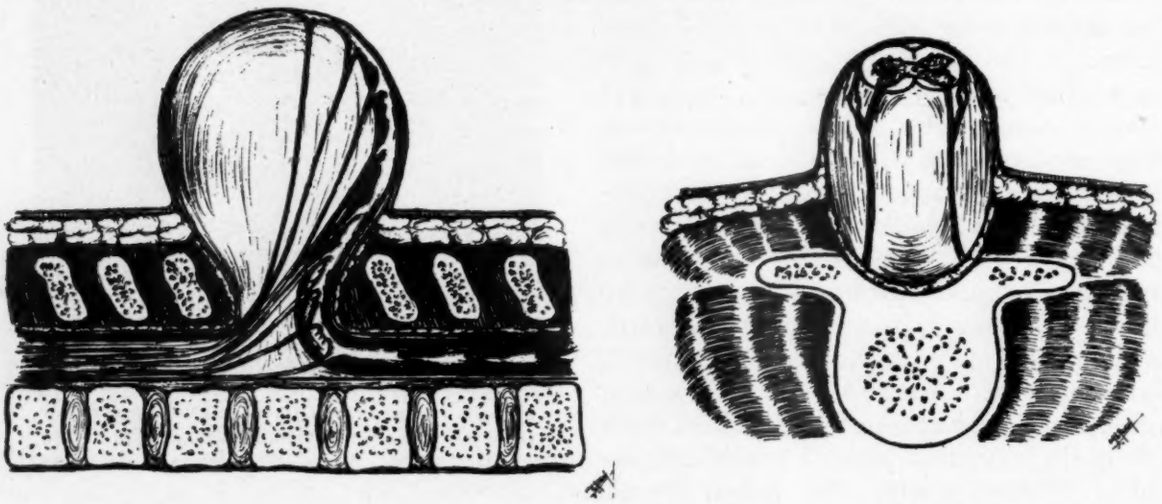
FIG. 12. D, the patient now aged nine years. The residual signs are occasional urinary incontinence and slight motor and sensory deficit in the lower extremities.

and cleansed with boric acid solution. The cilkloid is replaced as necessary, but not too frequently because of the deterrent effect of frequent changes upon epithelialization. When epithelialization is complete and adequate tissue for closure has developed, operation is considered. In the event of meningitis, surgery is deferred in favor of polyvalent chemotherapy which is instituted after taking cultures. The age of choice for operation is twelve to eighteen months unless circumstances force earlier intervention, at a time when growth of the child provides a stronger patient and lessens the operative risk. Operation is facilitated by more adequate tissue for repair and closure. In addition, intrinsic defects not detectable in the newborn come to light during these months of observation. Hydrocephalus or other outstanding deformities contraindicate operation. (Fig. 15.) A certain number of patients thought to be improper subjects for surgery as the result of hydrocephalus, neurologic deformity or questionable mental status may compensate in a two-year period.



A B

FIG. 13. Diagrammatic drawing of a meningocele. A, saggital section. B, coronal section.



A B

FIG. 14. Diagrammatic drawing of a myelomeningocele. A, saggital section. B, coronal section.

Therefore, it is a wise policy to give a guarded prognosis rather than an hopeless one until this time. During the period of trial and conservatism, most patients with myelomeningocele die of intercurrent respiratory tract infection, meningitis, ascending urinary sepsis or other maladies that result from concomitant malformation of other systems. The survivors of this group then present serious orthopedic problems. Plastic repair of the neural axial defect becomes necessary in order that appliances for the paraplegia may be worn with safety and comfort. Much of the orthopedic burden can be lightened by calling in the orthopedic consultant early in order that deformity by contracture may be avoided.

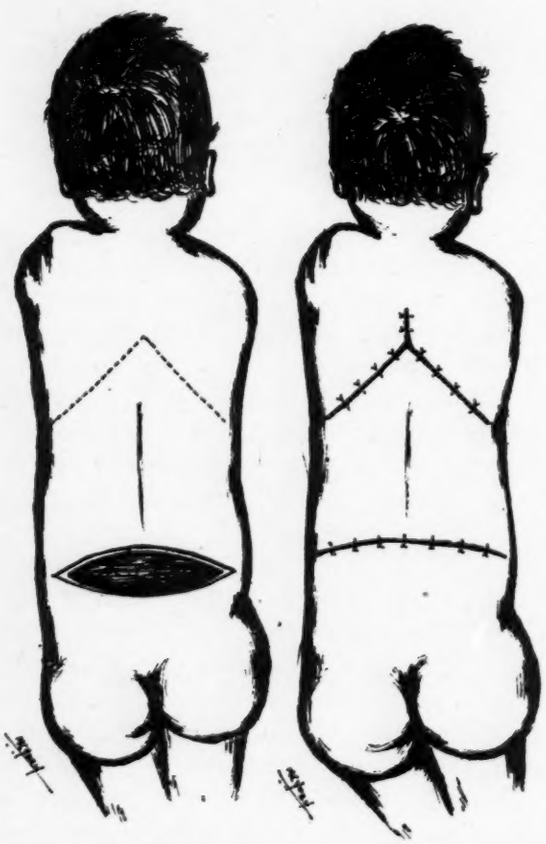
Preparation of an unusually wide field in operations for meningocele is necessary in many instances because of the frequent use of relaxing incisions to achieve closure without strain. The skin and protuberance may be prepared with any suitable chemical solution, preferably colored, in order to be certain that no area remains untouched. This is particularly important in view of the crevassing of these masses. It is well to have the patient's head slightly dependent throughout the procedure so that undue loss of spinal fluid by dependent drainage may be avoided. An elliptical incision running transversely to the neural axis and carried down to the deep fascia is usually more practicable than the vertical type. The skin and superficial fascia are mobilized by dissection up to the point of emergence of the neural sac from the bifid defect. Opening of the dura at a point obviously free from neural structure permits inspection of the contents with less chance of damage. At this point, a true distinction between meningocele and myelomeningocele is possible for the first time. Neural elements ending blindly in the wall of the sac are sacrificed, and presumably intact structures are returned to the neural canal. In resecting the sac, due care must be given to the preservation of sufficient dura for water-tight closure. Ingraham and Hamlin[25] advise that no attempt be made at bony reconstruction at this time. It may be feasible to reinforce the dural closure with flaps of fascia elevated from the sacrospinalis musculature, if in so doing, the procedure is not unduly prolonged and compression brought to bear on the contents of the reconstructed thecal sac. In large protuberances, in which the spinal canal is too small for the replaced neural elements, a reconstructed meningocele is created and relative safety achieved by closure of sound superficial fascia and skin above it. Not infrequently, a relaxing incision, such as the one Davis[26] attributed to Dieffenbach is useful in effecting closure without tension. (Fig. 16.)

FIG. 15. A and B, an inoperable lumbosacral myelomeningocele. Note relaxed rectal sphincter, deformity of the legs and neural elements presenting on the surface of the lesion.

Recently it has been the practice of this clinic to use a sulfonamide emulsion dressing surmounted by dry gauze and maintained by elastoplast adhesive. Lightweight rubber sheeting anchored by adhesive at the level of the apex of the intergluteal fold and carried forward over the entire dressing forms a further barrier against excretory contamination. The patient is

FIG. 16. A, photograph of a patient, eight months of age, with a large myelomeningocele.

moved from the operating table to a Bradford frame and secured in the prone position with padded restraints. A pliofilm covering of the frame prevents skin irritation. If there is any suspicion of incompetence of closure, the frame should be tipped slightly downward in order to reduce any hydrodynamic strain on the suture line. Vigilant nursing is required to observe cerebrospinal fluid leakage, decubitus formation and excretory contamination until the wound is thoroughly healed. Leakage occasionally occurs in spite of meticulous closure, possibly as the result of a change in cerebrospinal fluid dynamics created by operation. In this event, immediate corrective measures must be undertaken, such as resuturing of the wound at the site of fistula and relief of intracranial pressure by repeated ventricular taps. Should this not prove sufficient, a system of closed drainage of cerebral spinal fluid by either the lumbar or ventricular route is indicated. The pressure gradient can be controlled by a method described by Ingraham and Campbell.[27] Unless there is leakage or sepsis, the wound is best not examined until the eighth day. Sutures are usually removed between the eighth and tenth days. Urinary retention as a complication is handled by tidal drainage as advocated by Munro[28] with modifications made in the apparatus to adapt it to the age group in question.



B C

FIG. 16. B and C, diagrammatic drawings of the V-Y relaxing incision used to effect closure in this type of case.

CRANIUM BIFIDUM

Cranium bifidum corresponds with spina bifida and arises by the same process. Meningoceles occur in conjunction with cranium bifidum. Encephaloceles correspond with myelomeningoceles and arise as the result of faulty induction of ectoderm and mesenchyme.

INDICATIONS FOR SURGICAL INTERVENTION

Selection of patients for surgery in cranium bifidum and associated deformities is

A B

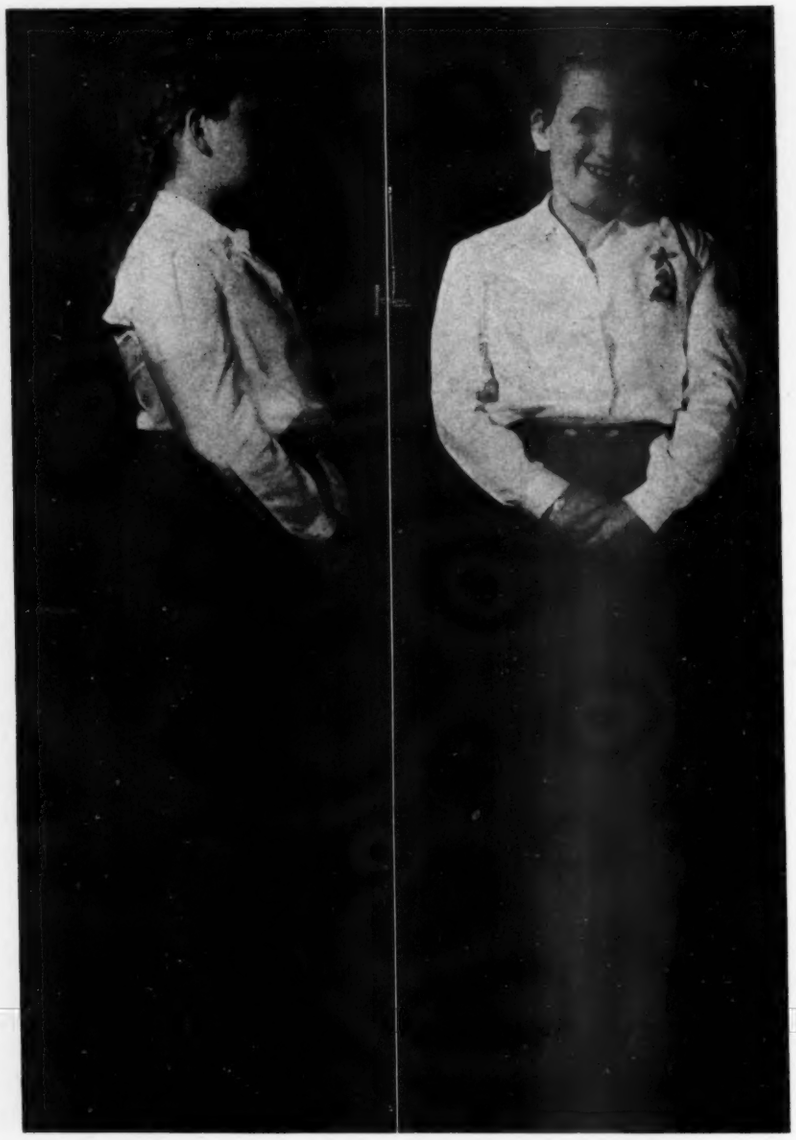
Fig. 17. These photographs are of the same patient: A, four and one-half weeks old; B, postoperative photograph at six and one-half weeks.

based upon the same considerations that were outlined for the spina bifida group. Abnormalities of neurulation form the embryologic basis for judging what patients are suitable for surgical readjustment of congenital malformation. In this way, operation will not be needlessly offered to those with uncontrollable hydrocephalus, feeblemindedness and blindness due to basic malformations of cerebral structures. The age of choice for operation is twelve to eighteen months, as is the case with the spinal group. However, earlier consideration must be given to individuals threatened by meningitis from rupture or incipient ulceration of the covering of the protrusion. If the infant appears to be normal in all other respects, it is worth chancing future development by operating as early as the first day of life. In general, any severe mental retardation in the first twelve to eighteen months of life contraindicates surgery, as does progressive hydrocephalus, blindness or signs of severe deformity in other systems. When the decision cannot be made by history, physical and neurologic examination, pneumoencephalography by the lumbar or ventricular route is indicated. Gross bizarre malformation of the intracranial structures, outlined by this means, will make the final contraindication.

Simple cranium bifidum is rare in this clinic. Ingraham and Swan,[21] in 1943, in reporting the site of eighty-four cases of encephalocele and cephalic meningocele showed that sixty-three of them were occipital (Fig. 17), as opposed to five nasal, six frontal, nine parietal and one nasopharyngeal. The higher rate of true encephalocele formation, as opposed to meningocele in the occipital region is interesting because this is the first part of the neural axis to close. It was also their experience that the prognosis was poorer when an encephalocele was encountered.

Surgery of simple cranium bifidum consists of plating the bony defect with tantalum in order to protect the underlying brain from direct trauma to which all children's skulls are subjected when they are learning to walk and play. (Fig. 18.) Fixation with tantalum wire rather than with pegs is desirable since the calvarium is so thin. For this same reason, an acrylic inlay is not advised.

Excision and repair of meningoceles or encephaloceles along the vault of the calvarium is not a difficult procedure if the position of the venous sinuses of the brain is constantly kept in mind. The incision



C D

FIG. 17. C and D, at eight and one-half years of age. The lesion removed at operation was a definite encephalocele which contained neural elements. This patient does reasonably well in school.

must be adapted to the position, size and shape of the defect in order that a water-tight dural closure may be effected and reinforced by firm galeal and skin closure. On the other hand, repair of some frontal (Fig. 19) and nasopharyngeal encephaloceles requires formidable intracranial surgery in order to effect an enduring water-tight closure in the region of the cribiform plate. Ingraham and Matson,[29] in 1943, described the successful handling of a case in detail. Dandy's description of the operative treatment of orbital meningocele published in 1929[30] is classic.

ARNOLD-CHIARI MALFORMATION

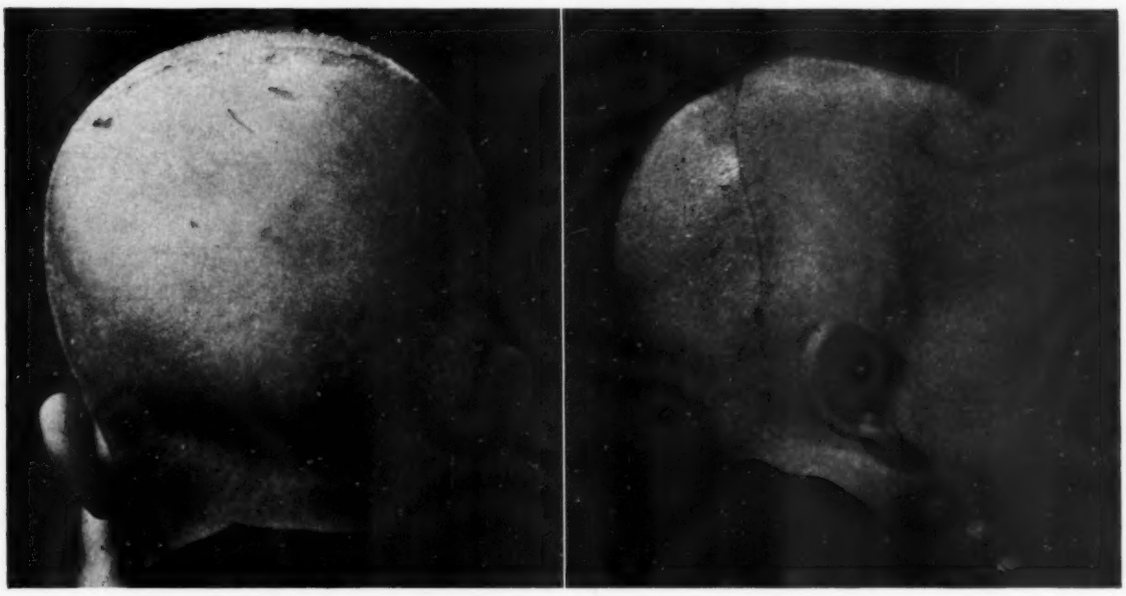
During the last twelve years, many excellent reports have appeared concerning the Arnold-Chiari malformation and its rôle in the production of hydrocephalus as a complication of meningocele and myelomeningocele formation. This discussion will not touch upon the origin of malformations such as Bucy and Lichtenstein[31] have re-

cently reported. Their patient showed no evidence of mesenchymal disturbance, such as platybasia or spina bifida.

In brief, the Arnold-Chiari malformation consists of downward displacement of the cerebellum and brain stem through the foramen magnum. Ascent of the cord occurs as the result of traction exerted by the relative fixation of the cerebellum and other components of the hindbrain within the calvarium, when the growth of the cord lags behind the more rapid growth of its supporting structure, the vertebral column. It is reasonable to suppose, therefore, that firmer fixation elsewhere along the neural axis, such as exists with meningocele or myelomeningocele might so reverse the direction of force exerted by the discrepancy of normal growth of the neural and spinal elements, that herniation of the posterior inferior portions of the cerebellum, hindbrain and adnexae results (Fig. 20), as well as reversal of the angle of the spinal nerve roots. (Fig. 21.) As this herniation is enhanced by growth, the constriction at the foramen magnum increases, with consequent obliteration of the subarachnoid space. Thus, passage of cerebrospinal fluid upward to the field of absorption in the neighborhood of the arachnoid villi is prevented. Russell and Donald[32] drew attention to the hindbrain malformation as a possible cause of internal hydrocephalus. These authors suggested that decompression of the spinal cord at the foramen magnum might lead to cure. Penfield and Coburn[33] three years later advanced the theory that fixation of the cord at the site of the bifid spinal column might produce

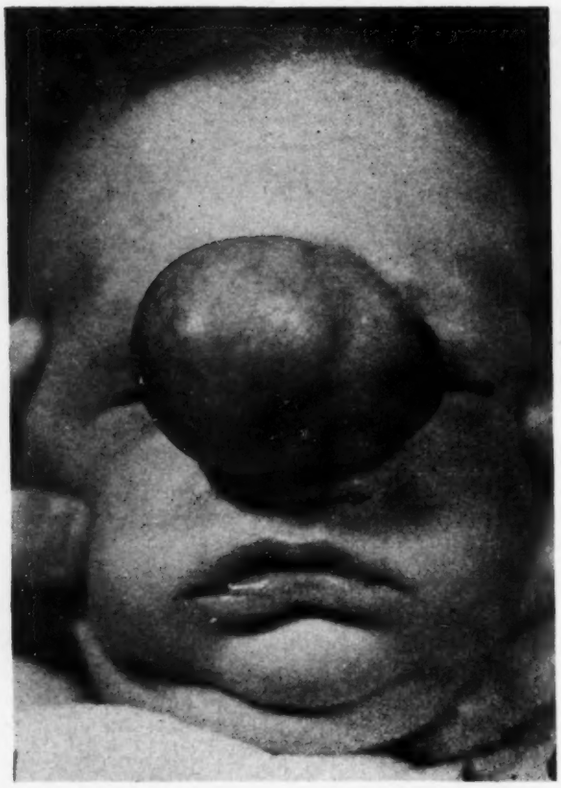
A

FIG. 18. A, photograph of a patient, aged one year, seven months, born with a simple cranium bifidum.



B C

FIG. 18. B and C, postoperative photographs after plating with tantalum.



A

B

FIG. 19. A and B, these photographs show a patient with a frontal encephalocele.

the malformation by traction on the hindbrain. List[34] raised the question whether a primary developmental deformity of the hindbrain would not better explain the structural aberration noted in many cases of the Arnold-Chiari malformation. List did not believe that the purely mechanical theory of origin, offered by Penfield and

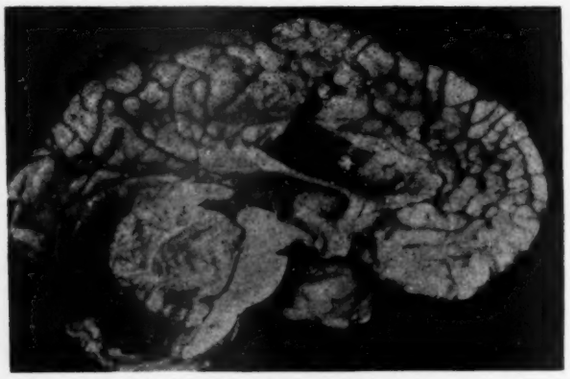


FIG. 20. Saggital section through the brain of a patient born with a myelomeningocele. Note the hydrocephalus, microgyria, and downward displacement of the inferior portion of the cerebellum and other hindbrain structures (Arnold-Chiari malformation).

Coburn, was applicable in all instances. Ingraham and Scott[35] reported the finding of microgyria in all of twenty cases studied in this clinic with Arnold-Chiari malformation associated with myelomeningocele. This finding is an important contribution to the list of other associated congenital malformations of ectodermal origin, such as stenosis of the Aqueduct of Sylvius, hypoplasia of the cerebellum, hydromyelia and diplomyelia. Associated defects of mesenchymal origin are frequently present in the upper cervical and basio-occipital region, such as fusion of cervical vertebrae (Klippel-Feil syndrome), spina bifida occulta, or platybasia of varying degree. Ingraham and Scott[35] suggested that microgyria in conjunction with myelomeningocele and the Arnold-Chiari malformation, constituted a clinical entity representative of a widespread defect. This is a tenable concept when consideration is given to the slight degree of differentiation of structures from which the cerebral hemispheres, cerebellum and fourth ventricle, are elaborated at the time that abnormal neurulation makes possible the formation of a low spinal myelomeningocele. In the late somite embryo, the anterior neuropore is closed on the twenty-fifth day, at which time the cerebral vesicles, primordia of the cerebellar hemispheres are but slight lateral dilatations of the prosencephalon. The fourth ventricle and primordium of the

cerebellum have not been formed at this time in the rhombencephalic region. Therefore, any change of sufficient magnitude to derange morphogenetic movement in the region of the blastopore with resulting abnormal neurulation and ultimate myelomeningocele formation at one point, may possibly interfere with induction elsewhere. Hence, microgyria of the cerebrum, hypoplasia of the cerebellum and other associated anomalies may be interpreted as manifestations of malinduction possibly resulting from interference with organizer substances of a higher degree, or the competence of tissue stimulated by them.

Lichtenstein[36] has shown that the Arnold-Chiari malformation may be a reasonable mechanical cause of hydrocephalus noted at birth in patients with myelomeningocele formation. If this occult deformity is present in the posterior fossa of a patient born with an apparently normal cerebral spinal fluid circulation, who, coincidentally, has a meningocele or myelomeningocele, it is likewise justifiable to suppose that it may be the cause of hydrocephalus occurring at any time after birth. The sudden onset of hydrocephalus following repair of a meningocele or myelomeningocele was attributed by Penfield and Cone[37] to loss of an absorptive mechanism contained within the sac. It, however, has been the experience of this clinic that preservation of the sac has not prevented hydrocephalus. The presence of the Arnold-Chiari malformation has been demonstrated repeatedly by this neurosurgical service in patients operated upon for meningocele or myelomeningocele who have developed acute hydrocephalus during their immediate post-operative course. Therefore, it seems reasonable to conclude that patients of this type possess a narrow margin of safety because of an extensive degree of posterior fossa deformity. The shift in cerebrospinal fluid dynamics caused at the time of opening the meningeal sac during operation may cause further downward displacement of the already herniated portions of the hindbrain. Under these circumstances, constriction of the subarachnoid space above the foramen magnum may be so firm as to block the ascent of fluid and cause hydrocephalus. Operation with the head slightly dependent is advisable on the grounds that it may prevent this chain of events. It follows then that posterior fossa exploration is indicated in patients who develop hydrocephalus at any time following repair of a meningocele or myelomeningocele, if the condition of the patient's extremities, sphincters and degree of hydrocephalus is

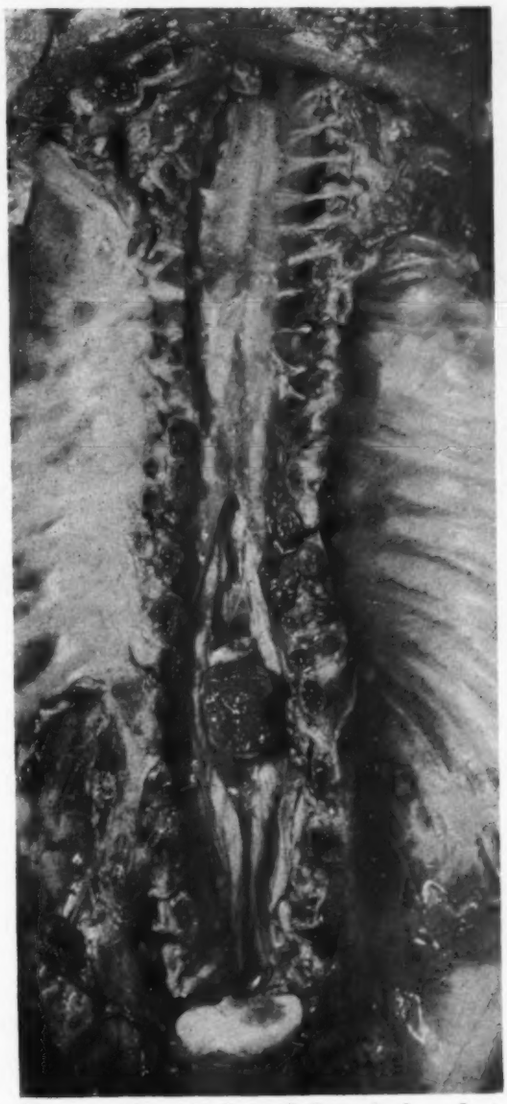


Fig. 21. Ventral view of the spinal cord at autopsy performed on a patient with an Arnold-Chiari malformation associated with a thoracic myelomeningocele. Note the upward course of the cervical nerve roots, diplomyelia and associated midline bony anomaly.

compatible with a relatively normal existence. Occasionally, a patient will be brought to the surgeon for the first time with a meningocele or myelomeningocele and hydrocephalus of such degree that, if abated, normal mental development might ensue. If the extremities and sphincters in such a patient are intact and the spinal lesion favorable for repair at a later date, posterior fossa exploration may be done prior to spinal surgery.

Exploration of the posterior fossa can be carried out through a midline incision. Wide excision of the occipital bone and the foramen magnum is important. The extent of cervical laminectomy is indicated by the lowest limit of herniation of the cerebellar tips. It is the experience of this clinic that anomalous venous channel formation in the suboccipital dura is as constant a finding in the operating room as microgyria in the postmortem room in patients with the Arnold-Chiari malformation. Therefore, it is advisable to exercise much care in the opening of the dura. Once the extent and nature of the anomaly has been estimated by exposure, adhesions are freed insofar as possible. If the herniated cerebellar tonsils are firmly fixed to the medulla and upper cervical cord, it is inadvisable to dissect them free, for fear of compromising the intrinsic circulation of these regions. Usually, lysis of adhesions on either side of the brain stem permits a free flow of spinal fluid from the region of the distorted fourth ventricle into the lateral recesses. However, if circulation cannot be re-established along the normal route, the by-passing procedure, originally described by Torkildsen,[38] for obstruction of the Aqueduct of Sylvius, may be employed. This consists of connecting the subarachnoid space of either lateral recess with the corresponding lateral ventricular system by means of a catheter. The catheter is first introduced into the lateral ventricle through a burr hole and then led subcutaneously to the lateral recess via the suboccipital decompression. Pains must be taken to see that the catheter is syphoning before establishing it in the subarachnoid space, lateral to the cerebellar hemisphere. When these procedures have been of no avail, partial extirpation and coagulation of the choroid plexus of each ventricle may be undertaken. Occasionally, this procedure, designed to reduce the production of cerebrospinal fluid, is sufficiently effective to bring about a balance between its absorption and production.

TERATOMA AND TERATOID FORMATIONS ALONG THE NEURAL AXIS

A sacrococcygeal teratoma is usually a large protuberant mass which greatly deforms the buttocks. Displacement of the orifices of the perineum may signal retrosacral extension of such degree that the bladder and rectosigmoid portion of the colon may be involved to the point of mechanical danger. Aside from the mechanical indications for operation, there is the constant threat of potential malignancy arising from epithelial elements developed within such structures. Lisco[39] in a review of the literature and report of two cases, points out that malignant degeneration in teratomas arising at this site, is as likely as elsewhere. MacCallum[40] believes that adenocarcinoma is the most common type of malignancy occurring in such lesions. This alone is an indication for early and complete resection, as has been the policy of this clinic for over thirty-one years.[41] Complete resection may usually be effected in one stage. When the lesion is very large in proportion to the total size of the patient and contains an extensive retrosacral component, it has been found practicable to carry out removal in two stages, the retrosacral dissection being undertaken as the second procedure. This permits use of the lithotomy position and greater ease in dissection in the retroperitoneal space. The actual surgical procedure is formidable only in that the mass is usually large in proportion to the patient, and liberally supplied by blood vessels. Varying degrees of spina bifida and meningocele formation accompany sacrococcygeal teratomas. Therefore, it is important to achieve a water-tight

dural closure and preservation of any herniated elements of the cauda equina. The decision for or against radiation should rest upon the result of a careful and searching pathologic study correlated with the completeness of the removal.

INTRACRANIAL AND INTRASPINAL TERATOMAS AND TERATOID TUMORS

Intracranial and intraspinal teratomas and teratoid tumors differ from the sacrococcygeal group in that they do not have constancy of position or develop, as a general rule, such a high degree of cellular differentiation. Craniopharyngiomas, pinealomas and chordomas will be omitted from this discussion because of the individual problems they present. The signs and symptoms produced by intracranial and intraspinal teratomas and teratoid tumors indicate the structures and levels involved. Ingraham and Bailey[16] conclude from a detailed study of a series of such tumors that congenital defects associated with signs of an expanding lesion suggest but do not firmly establish, the correct diagnosis preoperatively. Their study reveals that midline ectodermal defects occasionally overlie posterior fossa teratoid tumors, but more commonly exist in association with intrapsinal tumors. In some of their patients with this latter type of lesion, they were able to demonstrate roentgenographically a bifid defect. This was not their experience with the intracranial group.

SURGICAL MANAGEMENT

Exploration is indicated by signs of a space-occupying lesion. The decision as to the correct approach in the intracranial group rests with the correlation of data obtained by neurologic and roentgenographic examinations. Ventriculography is used, unless a dermal sinus points to the lesion, as has been the case with several patients operated upon in this clinic, who had teratoid lesions in the posterior fossa. The presence of spina bifida, with or without an associated ectodermal defect, correlated with neurologic and spinal fluid

FIG. 22. This photograph shows the surface opening of a persistent accessary neurenteric canal surrounded by a collar of abnormal fatty tissue in a child aged two years, eleven months.

studies, usually gives sufficient localization to permit exploration without myelography. The operative result and ultimate prognosis will be determined entirely by whether the lesion is discrete or infiltrative, composed of benign or malignant elements, and if it is so situated that removal can be effected without damage to surrounding neural structures. Malignant lesions can be dealt with only by decompression and radiation after biopsy. Certain benign intramedullary spinal lesions which would not permit complete resection because of their intimate application to the cord have, in Ingraham and Bailey's[16] experience, been benefited for more than five years by a partial resection and aspiration. Preoperative infection of teratoid lesions by bacterial invasion along an associated dermal sinus, greatly reduces the chances of a favorable outcome with a benign lesion. However, with the aid of such chemotherapeutic agents as sulfadiazine, streptomycin and penicillin, removal of an infected intraspinal dermoid has recently been accomplished in this clinic. Grievous past experience with atypical meningitis, de-

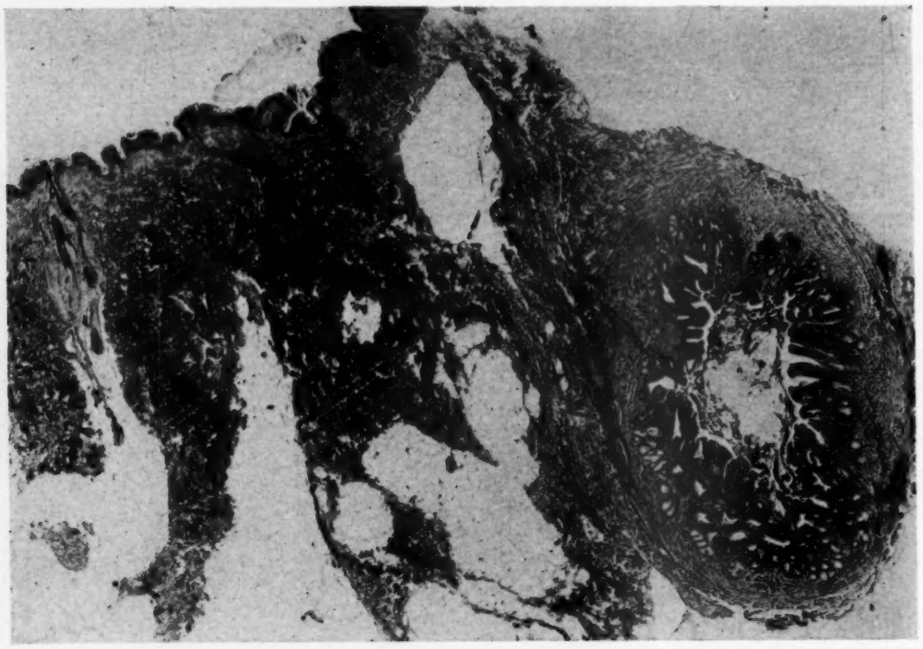


FIG. 23. A, this photomicrograph reveals the tract of the persistent accessory neurenteric canal surrounding skin and superficial fascia. Hematoxylin and eosin stain: magnification 15 diameters.

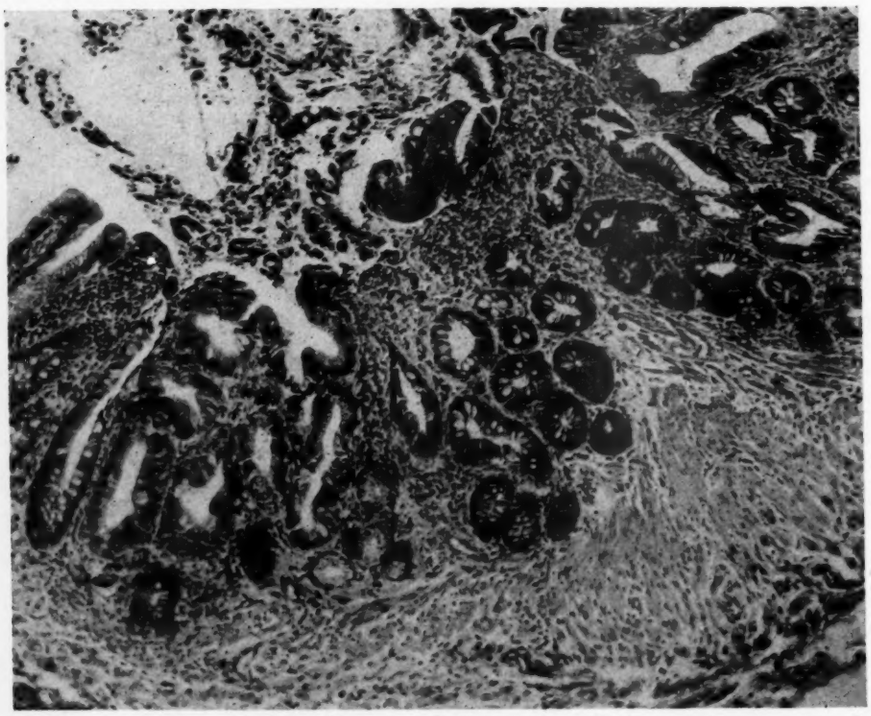


FIG. 23. B, further magnification, 130 diameters, reveals intestinal mucosa and muscular coat of the rudimentary intestinal tract.

veloping as the result of the extension of infection to the subarachnoid system from a septic teratoid lesion, has led to the policy that the presence of a midline dermal sinus, other than a pilonidal sinus, is sufficient indication for operating without waiting for neurologic signs to develop.

PERSISTENT REMNANTS OF THE NEURENTERIC CANAL

A theoretic basis for persistent neurenteric canal remnants and their association with spina bifida was proposed before in the basic embryologic discussion. This is an extremely rare condition in The Children's Hospital series (two cases). Spina bifida without neurologic deficit was present in both cases. The presence of an eccentrically placed fistulous tract (Fig. 22) in the lumbosacral region which might serve as a potential route for the spread of sepsis to subarachnoid system, was the indication for operation upon each patient. Operation consisted of block excision of the superficial ectodermal defect and surrounding skin, followed by mobilization of the tract down through the spina bifida to its point of emergence from the dura. In both patients a watery discharge from the fistulous opening onto the skin had been noted. In spite of this fact, no spinal fluid appeared on the transsected surface of the proximal stump of the tract. Nevertheless, a transfixing suture was placed about it to assure watertight closure of a potential connection to the subarachnoid space. It was then presumed that the watery discharge was a glandular secretion rather than cerebrospinal fluid. Both patients made an uneventful recovery. Microscopic examination of each specimen revealed normal skin and adnexae surrounding a tract lined by enteric mucosa, supported by muscularis and connective tissue elements, such as exist along the gastrointestinal tract. (Fig. 23.) These lesions may be teratomas, but it seems preferable to follow the hypothesis of Bremer[8] and to consider them as evidence of persistent neurenteric canal remnants displaced by an early aberrant morphogenetic movement.

SUMMARY

A review of relevent experimental embryology indicates that congenital anomalies of the neural axis are the result of disorders of neurulation. The surgical principles involved in the treatment of spina bifida, cranium bifidum and associated anomalies such as meningoceles, myelomeningoceles, encephalocles and the Arnold-Chiari malformation are discussed as well as teratomas and teratoid tumors arising along the neural axis. A correlation of embryologic and neurologic data is used in the selection of patients for surgery and the time for and type of operation to be performed on each individual case. The genetic basis of origin of congenital anomalies of the neural axis is considered as well as environmental factors that may cause disorders of neurulation. Hyperthermia in the mother occurring between the third and fifth week of gestation is suggested as a possible cause of such disorders.

The surgery of congenital anomalies can be more intelligently carried out when consideration is given to the embryology of the system concerned.

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CORTICAL EXTIRPATION IN THE TREATMENT OF INVOLUNTARY MOVEMENTS

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DURING the past fifteen years there has been an increasing interest and understanding of the problem of the surgical treatment of such abnormal involuntary movements as choreo-athetosis and the various tremors. Although Horsley[6] and others had operated upon a number of such patients many years before, interest in the problem had largely died down until the publication by Bucy and Buchanan[4] in 1932. In spite of numerous publications since then concerned with actual surgical procedures in human cases, animal experimentation and with theoretical considerations, the field is still one of active investigation, discussion and indecision. It is not surprising that this is so. Except for a very few instances involuntary movements comparable to those observed so commonly in human disease have not been produced in the experimental animal. Recent experiments by Ward, McCulloch and Magoun[8] have given cause to hope that this deficiency may soon be repaired at least in part. Nevertheless, this is still little more than a hope and thus far we have been dependent for our knowledge of the pathologic anatomy, pathologic physiology and the best means of therapy upon the study of those human experiments which nature provides for us. Suitable cases of this type are not too numerous and progress has been correspondingly slow. Thus far there is no surgical procedure which can be regarded as a satisfactory solution to the problems of these unfortunate people. *All operations thus far devised for the relief of abnormal involuntary movements must be regarded as distinctly experimental; all of them are unsatisfactory in major ways; no one of them has yet reached the stage where it can generally be recommended as a form of treatment routinely in any one of these conditions.* All operations for the relief of these conditions should be the occasion for intensive study. Much more knowledge is needed. Only by very critical, careful, objective observation and evaluation can we arrive at the true facts, the only sound basis for understanding the disease and planning intelligent therapy.

DEFINITIONS

The abnormal involuntary movements with which we are primarily concerned fall into two large groups: One of these is characterized by bizarre, irregular, unpredictable, non-repetitive types of movement which are commonly denominated by the term *choreo-athetosis*. These movements may be quick, slow or intermediate. Although they generally involve the peripheral part of the extremity more than the proximal, this is not invariably true; and in hemiballismus, which otherwise falls in this category, the movements are generally greater at the proximal joints than at the distal joints. The upper extremity is generally more severely involved than the lower but the degree of involvement is variable. In many instances the extremities are predominantly involved but in dystonia musculorum deformans or torsion dystonia, which is certainly a form of choreo-athetosis, there is generally marked involvement of the trunk as well. In general the movements are not only purposeless in fact but do not even have the appearance of purposeful movements. This is in contrast to the quick, jerky movements of Sydenham's chorea, which although involuntary, abnormal and purposeless so far as the individual is concerned, have, as Wilson repeatedly pointed out, the

appearance of purposeful movements. They are well coordinated muscular movements of the type which the patient might make voluntarily. This in general is not true of the movements of choreo-athetosis.

The second type of involuntary movement under consideration is the *tremors*. Although there are three different types of tremor which can be defined, they are not necessarily distinct entities. Many patients will present two or even all three types of tremor. Tremor may be divided into tremor at rest, static tremor and action tremor. Tremor is the involuntary occurrence of regular rhythmic movements in which the same movement is performed over and over again usually at a rate of from four to eight per second. *Tremor at rest* occurs in the supported extremity which is not involved by voluntary muscular contraction. Such tremors are characteristic of paralysis agitans or parkinsonism. It has been purposelessly argued that such tremors are not really tremor at rest as they are not present when the muscle is most completely at rest, i.e., during sleep. However, like all descriptive terms, this one has meaning which is capable of definition, of being readily understood by all who are familiar with the phenomenon and of being useful. The mere fact that the term cannot be stretched to cover some situation for which it was never intended is not an argument for introducing a new and unfamiliar term for one which is well established, or for denying the existence of the well recognized facts. The other two types of tremor might well be grouped together as intention tremors since both occur in muscle which is voluntarily innervated. The *static tremor* occurs in muscles which are engaged in supporting a part of the body stationary. *Action tremor* occurs in muscles which are engaged in moving a part of the body. Such tremors are very familiar by personal experience to most of us as they need not be dependent for their appearance upon the presence of organic disease. Both frequently occur as the result of a common functional disturbance, muscular fatigue. However, in the present discussion we are concerned with the more serious and persistent form of intention tremor which is characterized by actual alterations within the structure of the nervous system.

PATHOLOGY AND ETIOLOGY

These abnormalities of movement may arise as the result of various types of disease processes. Thus *infections* such as the infantile encephalitis may give rise to choreo-athetoid movements; the virus encephalitides may give rise to a parkinsonian tremor or to choreo-athetosis; syphilis of the mid-brain may produce the picture of paralysis agitans. *Developmental deficiencies* of the basal ganglia may produce athetosis or torsion spasm. *Degenerative disease* by affecting the same areas may produce the tremor both at rest and in connection with movement which is characteristic of Wilson's hepatolenticular degeneration. Degenerative processes may also lead to Parkinson's disease and to the bizarre movements of Huntington's chorea. *Vascular disease* by destroying various parts of the basal ganglia may produce tremor both at rest and in association with voluntary movement, may produce choreo-athetoid movements or the violent uncontrollable movements of the entire extremity which is characteristic of hemiballismus. Severe cerebral *trauma* may occasionally produce changes within the basal ganglia of such character and severity as to lead to the appearance of involuntary movements such as these. However, trauma is much less often responsible than these other disease processes. It is of interest, however, that neoplastic diseases in this region rarely produce any of these abnormal involuntary movements.

It is obvious from the foregoing that the nature of the causative disease process bears little if any relationship to the type of involuntary movements which develop. Rather the nature of the movements is largely dependent upon what part of the nervous system is destroyed, rather than

upon the nature of the destroying agent. Our knowledge in this field is not yet complete but in general the following statements are in accord with the findings of most investigators. *Tremor at rest*, or parkinsonian tremor, is found in association with destruction of the globus pallidus, the substantia nigra or both. It is surprising how frequently lesions are found in these two anatomically connected but separate parts of the basal ganglia in cases of parkinsonism. In this same connection it is of interest that in the monkeys in which Richter produced a tremor, as well as the rigidity and slowness of movement characteristic of parkinsonism, by chronic carbon disulfide intoxication, the essential and constant pathologic changes were in the globus pallidus and the substantia nigra. *Intention tremor*, on the other hand, is seen most consistently in association with lesions of the nerve fibers arising from the dentate nuclei of the cerebellum and passing forward through the red nuclei to the thalamus, whence their impulses are relayed to the precentral motor cortex. *Choreo-athetosis* is usually seen in association with lesions of the putamen and caudate nuclei of the basal ganglia but lesions only in the globus pallidus or in the thalamus have also been described. *Hemiballismus* arises as the result of destruction of the subthalamic body of Luys.

PATHOPHYSIOLOGY

It must be obvious to anyone who will give the matter a moment's thought that, although these abnormal involuntary movements may arise as the result of the destruction of these subcortical nuclei, they cannot be directly produced by these structures which are gone. The movements can arise only as the result of the activity of some portion of the nervous system which is still present and active. It has been quite commonly accepted that these movements represent the abnormal hyperactivity of some portion of the nervous system which has been released from the normal controlling influences of those portions of the nervous system which have been destroyed. We are indebted to the late Kinnier Wilson for having drawn our attention most forcefully to the probability that the precentral portion of the cerebral cortex and the motor pathways arising from them constitute the nervous mechanism which is released and which is directly responsible for the production of these movements. In 1942, I developed at some length a hypothesis explaining how each of these structures in the subcortical nuclei might exert a controlling, restraining, inhibitory or suppressor influence on the precentral motor cortex. This was based upon the known facts of neuroanatomy and neurophysiology, many of which had only recently been elucidated by the late Dusser de Barenne[5] and his disciples. No new developments since that time have brought forth any evidence to controvert that hypothesis. On the other hand, that hypothetical explanation is by no means the only possible pathophysiologic explanation of the manner in which the nervous system operates to bring about these abnormal movements. It is, however, the most complete explanation that has been proposed. I shall not here again present the details of that discussion. Suffice it to say that there are anatomic pathways leading from the cerebellum, substantia nigra, globus pallidus, putamen and caudate nucleus by way of the anterior part of the lateral nuclear mass of the thalamus to the precentral motor cortex of the cerebral hemisphere. There is also physiologic evidence that in some cases at least the functional activity of these pathways is one of suppression or inhibition of the activity of the precentral motor cortex. It has been my belief that these various subcortical neural mechanisms exert a controlling influence on the precentral motor cortex and that when that influence is removed the precentral cortex responds by an abnormal nervous hyperactivity which produces these involuntary movements. The type of movements which develop will depend upon the nature of the

controlling influence which is removed and will, therefore, vary depending upon which subcortical mechanism is destroyed.

The precentral motor cortex is a complex cortical area which has been fully discussed in the author's monograph.[2] The two principal subdivisions which are concerned with the innervation of the skeletal musculature and the production of movement are the two areas, designated as area 4 and area 6. The types of movement produced by these two areas differ in several respects. The various subcortical mechanisms mentioned above differ in their connections with the precentral motor cortex. It seems likely that some of them exert greater influence on one portion while others are more concerned with other parts of this cortical area. The precentral motor cortex gives rise to two large fiber systems. They are the pyramidal tract which extends directly from the precentral region to the spinal cord and the extrapyramidal systems which are much more complex and extend from the precentral cortex to subcortical areas, whence their impulses are relayed to other portions of the nervous system, including the spinal cord. There is some imperfect yet highly suggestive evidence that that portion of the pyramidal tract which arises from area 4 of the precentral cortex (some 50 per cent arises elsewhere) is concerned with the production of tremor, both at rest and in association with voluntary movement; while other evidence indicates that choreoathetosis is produced by impulses which travel over the extrapyramidal fibers which arise from both areas 4 and 6. As yet we do not know which of these various extrapyramidal systems is so concerned nor do we know which of the subcortical nuclear centers is connected with these extrapyramidal fibers.

SURGICAL TREATMENT

We are concerned here only with excision of the cerebral cortex in the relief of abnormal involuntary movements. Other surgical procedures such as section of the anterior limb of the internal capsule or extirpation of the caudate nucleus have been discussed elsewhere in this issue (see Browder).

In my experience it requires a much more extensive extirpation of the precentral motor cortex to abolish the movements of choreo-athetosis than it does those of tremor. This is not surprising if our hypothesis is correct that the movements of choreo-athetosis are produced by the extrapyramidal fibers which arise from both areas 4 and 6 whereas those of tremor are produced by pyramidal fibers arising only from area 4. At operation it is my practice first to determine the representation of the upper and lower extremities in the precentral region by stimulating the exposed cortex with a sixty-cycle sine-wave electrical current. Only when this has been carefully done can the surgeon plan his extirpation intelligently. Various persons have reported failures from what they claimed to be duplication of the procedure which I have advocated. Study of their operative reports or of the brains of their patients who have subsequently died has often shown that the extirpation was far too limited or that it was not placed in the precentral motor cortex at all. Such errors could not be made if the operator had carefully outlined the representation in this region by electrical stimulation.

In endeavoring to relieve choreo-athetosis, it is my practice to remove the precentral gyrus and part of the frontal gyri lying anterior to it. Posteriorly the extirpation should extend down to the bottom of the central or Rolandic fissure, usually a matter of 1.5 to 2.0 cm. Anterior to the central fissure the extirpation need not be so deep as it is necessary only to remove all of the grey matter down to the subjacent white matter. Any remaining fragments of grey matter situated in deep-lying fissures can be readily removed with the suction apparatus. It has been my experience that the extirpation is most easily carried out by a subpial dissection as advocated by Horsley and Sachs, using a curved semi-

sharp instrument such as a small periosteal elevator. In this way there is minimal damage to the vascular system and to neighboring gyri. Large arteries and veins crossing the site of the extirpation are easily spared, thus reducing the possibility of infarction of tissues at a distance from the extirpation. In most cases of choreo-athetosis both the arm and leg are involved. It is best, therefore, to remove the representation of both the upper and lower extremities. The extirpation should extend not only up to the interhemispheric fissure but over the crest of the hemisphere and down the medial surface to the first sulcus. In those cases in which the hemisphere is very adherent to the superior longitudinal sinus and efforts to separate them results in troublesome bleeding, this part of the extirpation, at the crest of the hemisphere, is best carried out with suction even though this destroys the tissue from this small area and precludes subsequent microscopic examination. In those few cases in which the choreo-athetoid movements are restricted to the upper extremity the extirpation can be limited to the related area and the representation of the lower extremity spared. Such a procedure gives rise to considerably less neurologic deficit, and thus the patient is less handicapped.

Obviously such an extirpation results in a partial paralysis of the contralateral extremities. As has been pointed out elsewhere (Bucy)[2] the resulting paralysis is not complete as the extremities receive some innervation from other parts of the brain, notably the remaining portions of the precentral motor cortex on the side of the operation and that of the opposite cerebral hemisphere. When it is possible to spare the representation of the lower extremity, the degree of paralysis in the arm is far less than when both the "arm" and "leg" areas are removed. When the representation of both extremities is destroyed voluntary movements in the upper extremity are severely limited. Little if any useful movement is present in the hand and fingers while the amount of movement increases proximally. In such cases the upper extremity is almost useless. For that reason no patient should be operated upon unless either the choreo-athetosis or the associated hemiparesis which so often accompanies it are so severe as to make the extremity practically useless prior to the operation. In every instance in which I have operated for the relief of choreo-athetosis the patient, both before and after the operation, has been happy to trade his uncontrollable involuntary movements for a greater degree of paralysis. No one of them has ever expressed any regret over his decision. It should ever be so.

Fortunately the degree of disability so far as the lower extremity is concerned is much less. This is true for two reasons. First the amount of bilateral representation in the cerebral hemispheres is greater for the lower than it is for the upper extremity. Therefore, the degree of paralysis which follows extirpation of the precentral motor cortex is less in the leg than it is in the arm. Second, the nature of the paralysis is such as to cause less disability in the leg than in the arm. As in the arm the paralysis is greater at the distal joints than it is at the proximal joints. Paralysis of the hand causes a much greater disability in a prehensile extremity like the upper one than does paralysis of the foot and ankle in a supporting member like the leg. Furthermore, as the paralysis is more or less spastic that, too, interferes with active function of the arm and hand whereas it is much less of a barrier to the proper function of the leg which is primarily concerned with serving as a rigid prop for the body. Although the upper extremity is rendered practically useless by such an operation I have never had a patient who was unable to walk following such an extirpation. Many of them are able to walk for miles and are engaged in occupations which require that they be on their feet a part of the time.

Because of the nature of the spastic paresis which of necessity follows this

operation, it of course is limited in its application to individuals who have choreo-athetosis on only one side of their body or in whom it is so much more severe on one side than on the other as to make a unilateral operation and unilateral relief worth while. The operation is not therefore suitable for cases of double athetosis or in cases of dystonia muculorum deformans. Furthermore, the operation does not attack the basic etiology or pathology of the disease and does not therefore have any effect in preventing progression of the disease or in relieving other manifestations of the disease. It should not be undertaken in cases in which the condition is obviously progressive or in which other symptoms are so severe and prominent as to make abolition of the involuntary movements of little value.

I should like to make it clear here that I have always found it most difficult to assess "improvement" in so far as mere lessening of the involuntary movements is concerned. The results of the operation described above are not mere improvement or reduction in the severity, extent or frequency of involuntary movements but the actual *abolition* of such movements. One should be content with no less. My observations would lead me to believe that where anything less than abolition has been obtained the operation has been incompletely or incorrectly performed.

As stated above my experience indicates that an extirpation limited to the precentral gyrus (area 4) suffices to abolish tremor. Again the extirpation should be performed subpially. It should extend, posteriorly, to the depth of the central fissure. It should include all of the representation of the arm or of the arm and leg depending upon the extent of the disease. Nothing in my experience leads me to believe that it is possible to abolish tremor by any procedure which does not interrupt the pyramidal tract or destroy the origin of that portion of it which arises from the precentral gyrus. At one time it appeared that an extirpation limited to the posterior one-half of the precentral gyrus (Bucy)[3] would suffice to abolish tremor. Such an extirpation removes the Betz cells from which the largest fibers of the pyramidal tract arise. Certainly such an extirpation has a profound effect upon the tremor but it will not abolish it completely and permanently.

Obviously even this limited operation results in a serious contralateral hemiparesis even though it is not quite as severe as that which follows the more extensive operation necessary to relieve the movements of choreo-athetosis. Therefore, it should not be undertaken unless the tremor is so severe that a hemiparesis will be welcome in its place. It must be borne in mind that here we are dealing with a profound difference between the two conditions. Choreo-athetosis is commonly associated with some degree of hemiparesis. Therefore, some increase in that condition, especially when it is attended with abolition of the very disturbing involuntary movements, hardly adds to the disability of the patient. With tremor the situation is different. It is rarely associated with any degree of paralysis of the affected extremity. The extremity is often a most useful member. One is, therefore, most hesitant to produce a paralysis where none exists. Only in the most aggravated cases should such a course be considered. Again, as with choreo-athetosis, the operation will not affect the progress of the disease. Most cases of parkinsonism are definitely of a progressive character and are, therefore, not suitable for such surgical treatment. What is gained by stopping the tremor in a man's left arm and rendering it useless, if next year or the year after the right arm will be similarly involved by tremor? It must also be remembered that the other symptoms of parkinsonism are not remedied by such an operation. In many cases the abnormalities of posture and of gait, the slowness and the poverty of movement, the muscular rigidity and the generalized aching are more annoying than the tremor itself. These symptoms will not

be relieved. In fact the rigidity will have added to it some measure of spasticity so that the stiffness of the extremities will most certainly be more marked after the operation than before. Furthermore, in my opinion the operation should be restricted to relatively young persons who are able to withstand the operation and, more important, are able to make the postoperative adjustments and to re-educate and develop the paretic extremities to the fullest possible degree.

I have tried to make it clear that in my opinion cortical extirpations, although of great interest in understanding the mechanism of tremor and of paralysis agitans, are of very limited usefulness in the treatment of parkinsonism. Only occasionally does one see a unilateral, non-progressive parkinsonism in a relatively young patient to whom the tremor is the greatest disability and for whom the tremor is so severe that both he and his physician consider it wise for him to exchange it for a partial paralysis of the affected extremities. Certainly not more than one in five hundred of the people afflicted with this disease could so qualify. Most of those for whom the operation is suitable are atypical cases, such as those post-traumatic and luetic cases that I have operated upon and reported.

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SECTION OF THE FIBERS OF THE ANTERIOR LIMB OF THE INTERNAL CAPSULE IN PARKINSONISM*

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IT seems reasonably well established that the tremor, and in some instances, the rigidity of Parkinson's disease may be favorably modified by (1) excision of a part of the precentral motor cortex, (2) division of a part of the anterior limb of the internal capsule, (3) section of the pallidofugal fibers or (4) section of the corticospinal fibers in the upper cervical cord. Removal of the head of the caudate nucleus on one side has no effect whatsoever on the tremor or rigidity of parkinsonism.

The present communication is primarily concerned with the technical features of the capsular operation and the results to be expected following section of an appropriate amount of the fibers coursing through the anterior limb of the internal capsule. This surgical procedure is still considered to be in the experimental stage and should be carried out only on a carefully selected group of patients with parkinsonism. If properly executed, the tremor can be abolished without paralysis. Moreover, the results following this operation are superior to those that I have had the opportunity to observe in patients submitted to any of the other three above enumerated procedures.

CAPSULAR OPERATION FOR PARKINSON'S DISEASE

The operation is conducted under local anesthesia without pre-operative medication. The subject is placed supine on the table and the drapes so arranged to give a clear view of the extremities contralateral to the side of the brain to be surgically exposed. These extremities should not be restrained or otherwise encumbered, especially the upper one. A small bone flap is outlined and turned down in the midfrontal region—fashioned to expose the falx and the superior part of the frontal lobe. After reflecting the dura and gaining proper orientation, a 2.5 to 3 cm. incision is made in the cortex just anterior to the premotor area and 3 cm. from the falx which it parallels. (Until well acquainted with the approach to the lateral ventricle it is well to locate the anterior aspect of the ventricle with a cannula which is left in place as a guide.) The incision is carried into the most anterior aspect of the lateral ventricle, thus exposing the head of the caudate nucleus. (Fig. 1.) The upper half of this structure is removed either by suction or scoop, thereby exposing the inner fibers of the anterior limb of the internal capsule. (Fig. 2.) The most anterior of these fibers in the corona radiata curve in rainbow fashion from forward aft to enter the capsule. Using a blunt right angle hook, the arm of the hook being 0.8 cm. in length, section is begun at the rostral end of the capsule and at a level just short of the termination of the corona radiata. (Fig. 3.) (A medium-sized blunt-end probe may be appropriately bent and used for sectioning.) Until familiarity with the operation is attained, it is best to section about 1 mm. of the capsular fibers at a time, then wait two or three minutes. During this rest period the surgeon himself makes observations regarding the amplitude of the tremor of the contralateral hand and the grip power is recorded. Slowly, bit by bit, the fibers are sectioned toward the knee of the capsule and observations as suggested are made and

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recorded. As a point approximately 1.5 cm. anterior to the knee of the capsule is approached the patient not infrequently becomes difficult to arouse and only after rough prodding is he capable of cooperating. After five to ten minutes the drowsy state passes and the tremor which usually abates during the drowsy period again becomes active. Further sectioning of the capsular fibers up to a point approximately 1 cm. from the knee completely abolishes the tremor. This is not the end point of the operation. If further sectioning is not carried out, the tremor usually returns within a few weeks after operation. It is therefore important to carry the sectioning further and the operation is completed only after the production of a marked paresis of the hand. (Fig. 4.) In other words, the patient is just able to elevate the upper extremity

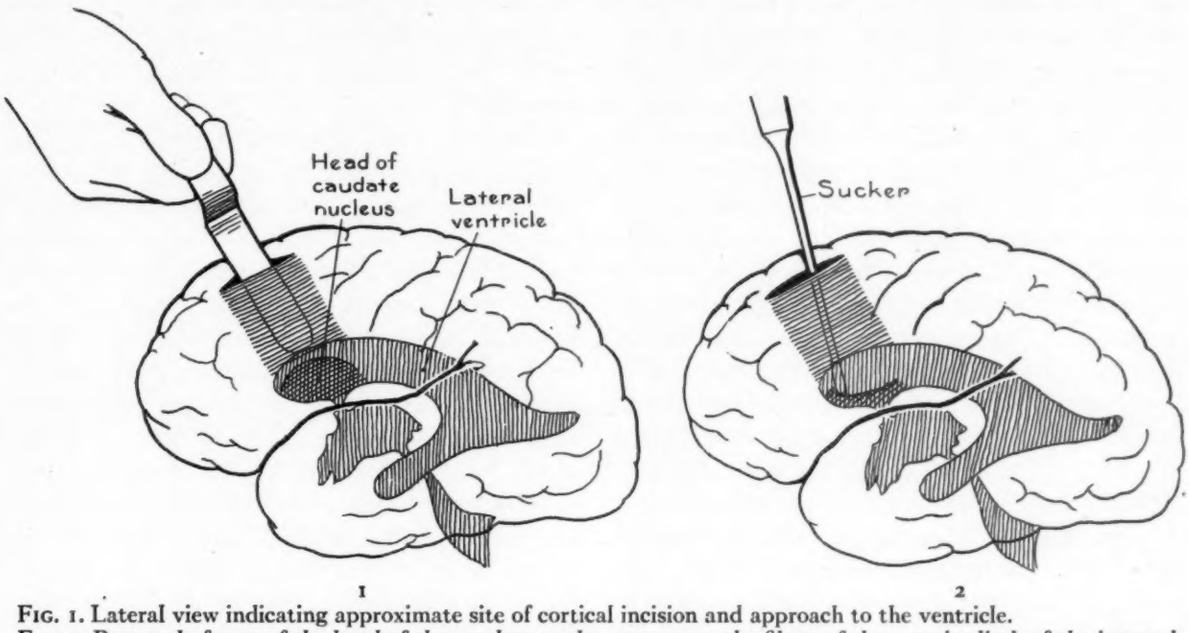

Fig. 1. Lateral view indicating approximate site of cortical incision and approach to the ventricle.
Fig. 2. Removal of part of the head of the caudate nucleus to expose the fibers of the anterior limb of the internal capsule.

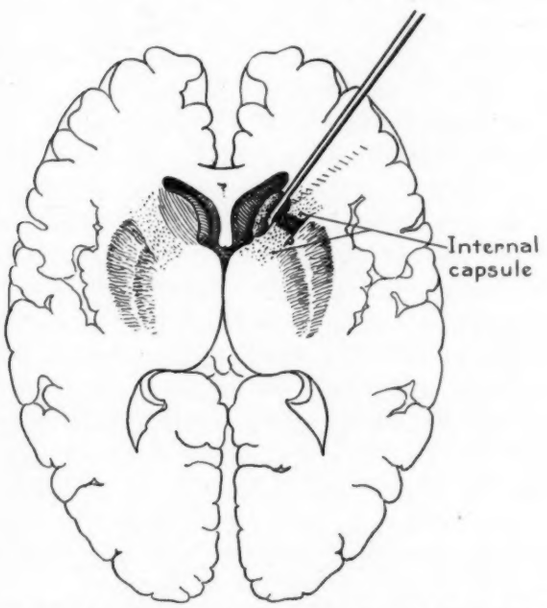

Fig. 3. Manner of sectioning the fibers of the internal capsule.

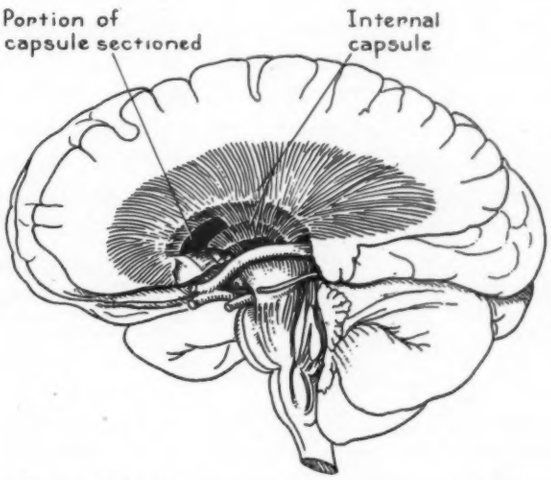

Fig. 4. Indicating approximate amount of fibers to be sectioned.

from his side and is only capable of slight flexion of the fingers without being able to grip the observer's hand. Examination of the lower extremity at this time will show that this part is equally paretic with the upper. Following complete hemostasis the wound is closed in the usual manner with layer silk. Immediately after operation the extremities affected by the procedure are relatively flaccid, paretic and Babinski's sign and the so-called confirmatories are present. The abdominal reflexes are abolished on the involved side. The patient is usually drowsy but easily aroused and cooperative. On the day following operation the hemiparesis is often more severe, gross movements of the involved parts, however, being preserved. There is a tendency for the patient to "sleep" when left undisturbed. Food and fluids are taken readily. Excessive sweating may be evident. A mild degree of fever prevails but hyperthermic states are uncommon. Gradually the motor power returns and by the tenth to twelfth day, when the patient is allowed out of bed, a mild to moderate hemiparesis is demonstrable. Slowly, ability to perform skilled acts with the paretic hand is regained. Forced grasping has not been observed.

COMMENTS

Inasmuch as the known facts regarding the etiology, pathophysiology and clinical course of Parkinson's disease have been recorded elsewhere by numerous authors, it seems unnecessary to restate the old story here. Suffice it to say that the abnormal neural mechanism that produces tremor and rigidity is not known and that all the explanations for these features of the disease must as yet be considered wholly philosophical. It is well known, however, that the disease advances or at least the disability resulting from tremor and rigidity is usually progressive and that medicinal therapy has not been very effective. For these reasons and others, it has been entirely proper to explore the possibilities of surgical therapy for those grossly incapacitated or in some instances for those who have completely withdrawn from society because of the unsightliness of their affliction. Certainly any patient with parkinsonism who is capable of continuing with his occupation, although somewhat handicapped, should be encouraged to do so and not to be imbued with the notion that an operation will restore him to normal.

There is a group of patients with parkinsonism who are relatively young but some have no gainful occupation principally because they are unable to obtain employment. Often after an unsuccessful struggle for a livelihood they are eventually incarcerated in a municipal institution. Here because of physical inactivity they rather rapidly become worse, especially as regards rigidity. Others are compelled to give up their jobs because of the incompatibility of the gross tremor and the technical skill required of them in their work. Still others, particularly young women, seclude themselves in their homes, unwilling to undergo the embarrassment attending average social activity. Among all of these there are some who may be restored to usefulness. The patients selected for operation should be under fifty years of age and not badly deteriorated physically. Tremor should be the outstanding feature of the disease and this for the most part limited to the extremities of one side. The patient and his family should have a complete understanding concerning the operation and what the outcome, barring complications, will be. They should be appraised of the future course of the disease and that surgical efforts will not, so far as is known, favorably influence the slow, down-hill course of the disorder. In short, the surgeon is operating for tremor and if, after careful assessment of all factors, it is thought that by abolishing the tremor the patient's life would be happier and the possibility of restoring him to gainful employment seems great, the operation should be carried out.

As stated, the average patient who has been submitted to section of the fibers of

the anterior limb of the internal capsule is able to walk, but with a mild limp, within two to two and a half weeks after operation. In some there is still a detectible residual slight limp after several months, however, this is scarcely noticeable and certainly not incapacitating. Gross purposeful movements of the paretic upper extremity return within ten days to two weeks after operation and during the ensuing month finer finger and hand movements are acquired. In fact, greater facility in performing skilled acts is often evident as contrasted with the pre-operative function. Frequently the patient will state that the affected hand is "normal," however by tests a mild dyspraxia can be demonstrated. If the tremor is abolished as described and a very slight hemiparesis endures, the possibility of a recurrence of the tremor is remote. Under unusual emotional stress transitory bursts of tremor have been observed in some, but these spells have been of short duration. Even though a very satisfactory result as regards tremor has been attained, in some patients the rigidity component while favorably modified is seldom completely abolished. If the proper selection for operation is made, namely, patients with gross alternating tremor limited for the most part to the extremities of one side, then rigidity is not a prominent feature. Marked rigidity and gross tremors do not co-exist. Recent experiences seem to indicate that rigidity may be favorably modified by an additional maneuver while performing the capsular fiber section; however, this of necessity will be subjected to further observations before conclusions are reached. It has been thought by several members of our staff that the so-called masked facies so characteristic of parkinsonism is less evident after operation. Possibly so, but certainly the blank, expressionless facies is not abolished. The same may be said of the greasy, sweaty skin. In many, it seems that this feature is less in evidence, however, no estimations have been made with precision. There is no doubt that the general appearance of the patients on whom successful abolition of tremor has been accomplished is markedly improved. They stand more erect, walk with a "positive" stride and will smile under appropriate stimulus.

Of the twenty-seven patients with parkinsonism submitted to operation, fifteen had the procedure performed as herein described. Nine of the fifteen had as the outstanding manifestations of the disease gross alternating tremor limited for the most part to the extremities of one side. The tremor was abolished by operating in six, absent for the most part in one and diminished for six weeks to two months in two patients. The remaining six patients had involvement of all extremities, about equal bilaterally. In three of these the rigidity component was the principal feature, in fact they were designated as "frozen." All three of these died as a result of the operation: one on the fourth, one on the eighth and one on the eleventh postoperative day. Autopsies were carried out on two, however, the examinations failed to disclose any obvious cause for the deaths. The other three patients of the group of six had gross bilateral tremors, about equal on the two sides. Although unilateral operations produced a cessation of tremor on one side and this without paralysis, it seems questionable whether or not the situation as a whole has been improved in any of them.

One patient in the group of fifteen, upon whom the capsular operation was since performed, has had four generalized convulsions during the past five years. These attacks have not incapacitated him since. He has been employed as a watch repairman for the past three years, has had no tremor and says his hand is "normal." An additional two patients have also reached the five-year period since operation without recurrence of tremor. Moreover, no tremor has appeared in the extremities of the "uninvolved" side in any of these three.

The capsular operation has not been carried out bilaterally by me but Hamby has had experiences with three patients with alternating tremors upon whom he performed section of the capsular fibers on both sides. From his personal communication it would seem that the results were not good and that this particular procedure should be carried out only on one side.

As yet the operation has not been sufficiently perfected for me to produce an enduring cessation of tremor in all cases; however, more recent results indicate that one should be able to abolish tremor in a high percentage of patients selected as described. Certainly the results are sufficiently good to warrant continued efforts.

TRIGEMINAL neuralgia or trigeminal tic douloureux is referred to by various other names, including major trigeminal neuralgia and trifacial neuralgia. It is characterized by brief attacks of excruciating pain in the face.

The brief excerpts in this issue have been taken from "Surgical Treatment of the Nervous System" edited by Frederic W. Bancroft and Cobb Pilcher (J. B. Lippincott Company).

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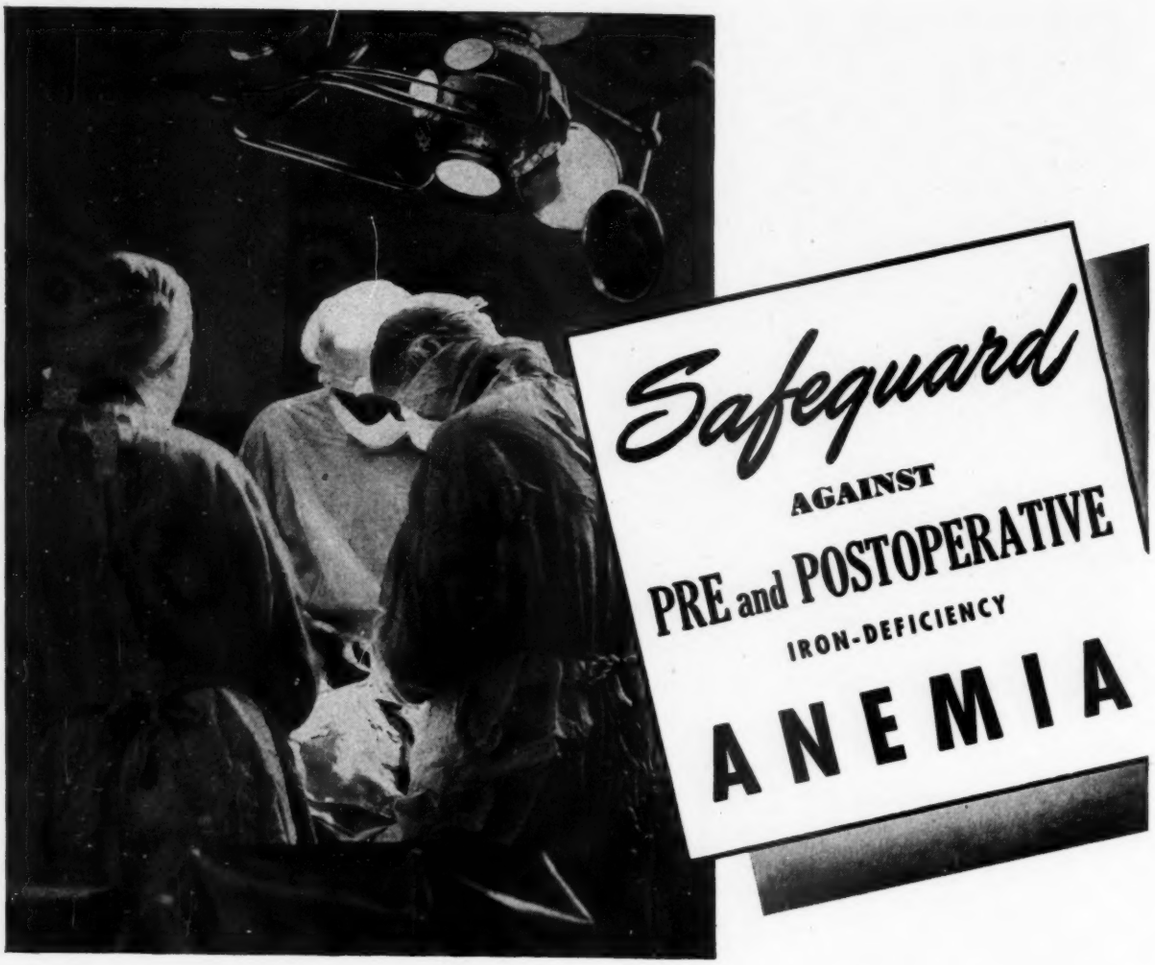

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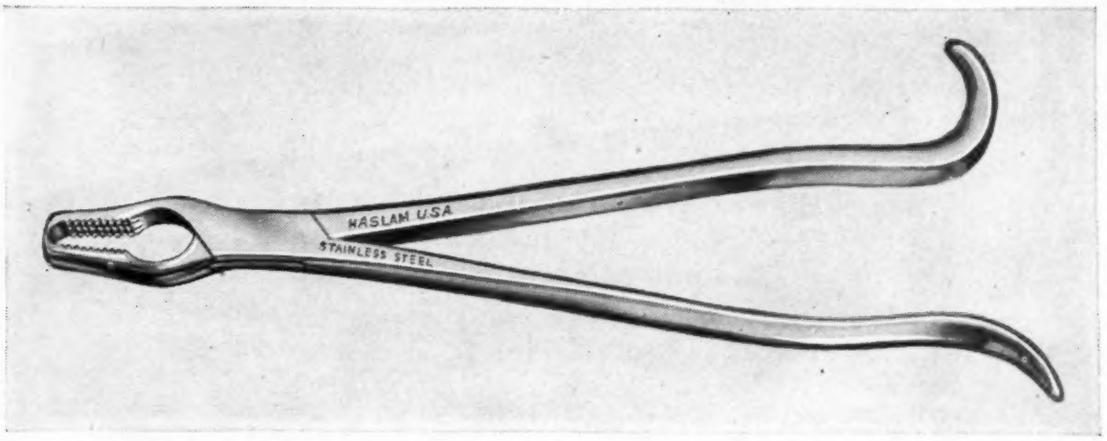

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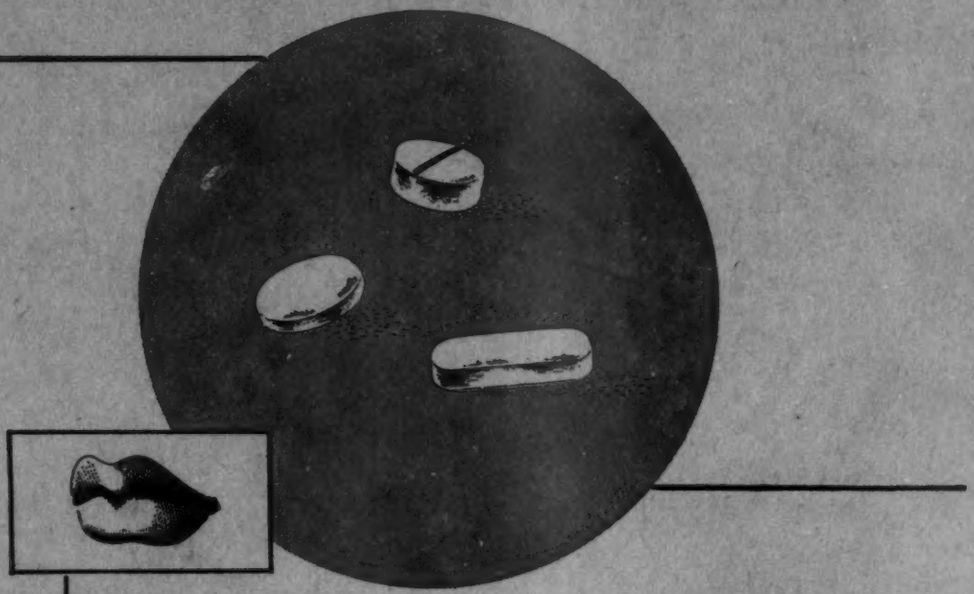

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